Essays in Behavioral and Development Economics

Dissertation for the Faculty of Economics, Business Administration
and Information Technology
of the University of Zurich

to achieve the title of
Doctor of Philosophy
in Economcis

presented by

Johannes Haushofer
from Germany

Approved in April 2012 at the request of
Prof. Dr. Ernst Fehr
Prof. Dr. David Laibson

The Faculty of Economics, Business Administration and Information Technology of the University of Zurich hereby authorizes the printing of this Doctoral Thesis, without thereby giving any opinion on the views contained therein.

Zurich, April 4, 2012

Chairman of the Doctoral Committee: Prof. Dr. Dieter Pfaff

Abstract

This thesis asks two broad questions: first, what are the neurobiological and psychological consequences of poverty? Second, do these consequences, in turn, influence economic behavior, and could these influences perpetuate poverty? The findings presented here speak to a particular incarnation of these two questions: first, does poverty cause stress? Second, does stress lead to short-sighted economic decisions, which then exacerbate poverty? I address these questions in five studies.

First, in a laboratory experiment in Switzerland, I find a strong correlation between socio-economic status and baseline cortisol: a 1% increase in income is associated with a 3% decrease in baseline cortisol levels, even after controlling for a variety of other socioeconomic factors. However, a crucial question is whether this relationship is causal.

In the second study, I address this question using two natural experiments in Kenya: in a region populated by Maasai tribespeople, I study the effect of a drought on stress levels. I find that the proportion of livestock lost by each household is not predicted by any observable variables, making the loss of livestock a random negative income shock. I find that a year later, baseline cortisol is significantly higher in families who lost more livestock than others. Second, in a farming district on the slopes of Mt. Kenya, I use rainfall variation as an exogenous source of income variation to identify an effect of income shocks on stress levels. I find that periods of no rain lead to significant increases in salivary cortisol, with a lag of 10 days. Thus, exogenous increases in poverty lead to increases in salivary cortisol, establishing a causal link between poverty and stress hormones.

Third, I study the causal effect of poverty on other psychological variables. Using data from 60,000 households in 41 countries, I find a strong relationship between income and locus of control (LOC), i.e. the degree to which respondents feel that they control their lives vs. that external events determine their fate. This relationship holds both within and across countries, and is robust to controlling for a number of socioeconomic covariates. More importantly, I establish a causal link from income to LOC using the average distance of a country from icefree coasts as in instrument for GDP. Thus, poverty not only affects stress, but also locus of control – an important variable both because of it affects economic productivity, and because it is closely related to mental health and depression.

Fourth, I conduct a laboratory experiment in which I ask if I ask whether negative income shocks have consequences for economic choice. Subjects perform an effort task from which they can earn income. A random subset of subjects then receives a negative income shock. I then ask whether this

shock has consequences for economci behavior, and find that subjects who receive the negative income shock show increased levels of present bias. This effect is not observed in subjects who received a *positive* income shock, nor for a control group which has the same level of wealth but did not suffer an income shock. Thus, negative income shocks have a specific effect on economic behavior in that they increase present bias.

Finally, I ask whether stress causally affects economic choice, in particular, time preference. Using the Trier Social Stress Test (TSST) to induce stress in the laboratory, I find two dissociations in the effect of stress on time preference: first, I experimentally and econometrically distinguish between two aspects of discounting: impatience, captured by an exponential discount function, and present bias, captured by a quasi-hyperbolic discount function. I find that stress affects present bias, but not impatience. Second, immediately after stress, stressed subjects are more present-biased than controls, while 20 minutes later ("late" group), they are less present-biased than controls; this result is consistent with a rich literature documenting an inverted-U relationship between stress and performance. The magnitude of the behavioral change within the stress group is correlated with the magnitude of the stress-induced increase in cortisol, suggesting that the effect may be driven by corticosteroids.

My results suggest that poverty may cause increased levels of stress, in particular the stress hormone cortisol; and that increases in stress, in turn, can lead to short-sighted economic choice. Together, these findings suggest that poverty could perpetuate itself through neurobiological and psychological "vicious circles", in which poverty has particular psychological and neurobiological consequences, which in turn lead to behaviors that reinforce poverty. These factors could offer new possibilities for poverty alleviation in the future, and thus bring us a step closer towards solving this lingering global problem.

Acknowledgements

This dissertation is a beginning more than an endpoint; it marks my first few steps within behavioral and development economics, and I hope that many more will follow. The three years during which I developed the ideas and studies presented here were enormously enjoyable and rewarding, and heartfelt thanks go to all the people who have made them so.

First and foremost, my thanks go to my advisor, Ernst Fehr. He took an interest in my ideas at the intersection of psychology, neuroscience, and behavioral and development economics at a time when they were so immature that they shouldn't have taken up anyone's time, much less his. He offered me the wonderful opportunity to pursue my PhD in the unique research environment he has created in Zurich. Most of all, he supported my unorthodox research interests with unwavering enthusiasm, and endured with endless patience my Subsaharan escapades and the circuitous paths I took to find answers to my questions. The breadth of his vision is truly unique not only in economics, but in science in general, and I cannot thank him enough for the generosity with which he shared it with me. Thank you Ernst!

My second examiner, David Laibson, has been much more than a second examiner: like Ernst, he supported my resaerch agenda from its very beginning, and has been enormously generous with his scarce time and sage advice. His openness to ideas and concern for students' interests is an inspiration, and I am very grateful for it.

While Ernst taught me to walk in behavioral economics, others did the same for development economics: I am deeply indebted to Tavneet Suri, Joost de Laat, Matthieu Chemin, and Caroline Archambault for the openness with which they welcomed me into their field, and for the generosity and insanity which it must have taken to let me manage my first randomized controlled trial. Jeremy Shapiro and Paul Niehaus deserve the same credit for taking me onboard their pathbreaking work on unconditional cash transfers in Kenya. I am greateful to Oeindrila Dube and Bilal Siddiqi for the very enjoyable collaboration on incentives for health workers in Sierra Leone, and for Barbara Lohman-Payne for the excellent collaboration on immunological outcomes in Kenya.

Both in Kenya and Switzerland, Tobias Kalenscher has been a wonderful friend, colleague, and collaborator; I am continuously delighted over by the similarity of our thought process, and look forward to many more years of rewarding collaboration. It has been a privilege to collarborate with Sandra Cornelisse, Marian Joëls, and Daniel Schunk on lab experiments in Zurich. I am particularly grateful to Sandra and Marian for sharing their expertise on stress, for tolerating my outlandish questions in this domain, and for

their indredibly hard work during the publication process; and to Daniel for his upbeat and positive outlook and his econometric widsom. Being Christoph Eisenegger's coauthor has been a great experience, not least in terms of benefiting from his encyclopedic knowledge. I thank my neuroscience PhD advisors: Marge Livingstone and Nancy Kanwisher, for their continued generous support and good spirit; Nancy in particular continues to be my scientific role model and a wonderful mentor and friend.

My work both in the field and the lab has been made possible by an outstanding group of administrators and research assistants. I am grateful to IPA Kenya's country director, Colin Christensen; my research assistants Matthew White, Marie Collins, Faizan Diwan, Amos Odero, Benedetta Mwongeli, Joseph Njoroge, Averie Baird, Ellen Moscoe, Jennie Glassco, and Harrison Pollock; to IPA Kenya's excellent team, especially Carolyn Nekesa, Adina Rom, Daniele Ressler, and Febrone Atieno; and to my friend Gesche Hübner for her contribution to our fieldwork. Karen Levy has been a constant source of inspiration and support for my work in Kenya, and I am deeply indebted to her. In Zürich, I thank Ursi Meier, Bettina Petralli, Stefan Schmid, and Regina Notz for their excellent administrative support, and Nina Spiri, Franziska Föllmi, Matthias Hartmann, and Claudia Paixao for support with running experiments. I am particularly grateful to Helen Bernhard and Sally Gschwend for support not only in administrative, but also all other conceivable matters. I thank my Bachelor's and Masters thesis and seminar students, Florian Bosshart, Alain Kamm, Deborah Kistler, Maayke Seinstra, Roni Madar, and Amelie Brune, for their hard work and the rewarding collaboration. This dissertation uses a LyX template produced by Daniel Mettler, whom I thank for making it available.

It turns out, fieldwork is expensive. I am grateful to the Science of Behavior Change Initiative at the NIH, in particular Jonathan King and Richard Suzman, and the Cogito Foundation, in particular Irene Aegerter, for singularly generous support of my high-risk research agenda.

During the last stages of my Economcis PhD, I have been incredibly fortunate to find a new home in Harvard's program of Prize Fellowships in Economics, History and Politics and the Jameel Poverty Action Lab at MIT. Having the opportunity to work in this environment has been the fulfillment of a dream, and reality has lived up to every aspect of it. For making this experience possible, I am deeply grateful to the Senior Fellows of the Program in Economics, History, and Politics: Allan Brandt, Walter Johnson, Richard Tuck, Amartya Sen, and in particular, Emma Rothschild and Abhijit Banerjee; and to the directors of J-PAL, Esther Duflo, Abhijit Banerjee, and Rachel Glennerster. I am particularly grateful to Esther and Drazen Prelec for first conspiring to bring me to J-PAL. My fellow fellows, Ben Golub,

Rachel Leow, Noah Millstone, Alexandra Yates, and Valerie Wallace, are a wonderful bunch whose friendship and stimulating conversations I cherish.

The years of my PhD have not only been scientifically exciting, but also full of wonderful friendships. First and foremost, I thank Benjamin Vogt, Pietrino Cadoni, Karen Levy, Nancy Kanwisher, Lina Nerlander, Laura Deneke, Petra Persson, and my sister Lisa for their wonderful friendship, warmth, and love; after two PhDs I still haven't understood how I deserve them. I am grateful to Pierre Stallforth, Susan Joho, Camille Doykan, Ryan McKay, Molly Crockett, Dina Pomeranz, David Seim, Pavan Ramdya, Suliana Manley, and their daughter Nalani, for making my days brighter; and to the members of Nota Bene Zurich and the Boston Choral Ensemble for memorable musical moments.

Finally, I thank my family, Ingrid, Gotthard, and Lisa, for being the pillars of my life; they give me roots and wings, and my gratitude to them cannot be expressed in words. This dissertation is dedicated to my beloved sister Lisa, whose wonderfulness I am only beginning to understand, and whom I hope to convince one day of how amazing she really is.

Contents

Part I

The Behavioral Economics and Neurobiology of Poverty

Chapter 1

Introduction

The motivation for the research described in this thesis departs from the simple question: What scientific problem should I be working on? I.e., which is the most pressing question facing the world today?

In my view, there is an elephant in the room: one billion people worldwide still live in abject poverty. Arguably, many other problems – e.g. disease and violence – are at least partly consequences of this fact. In the research presented here, I integrate approaches from neurobiology, psychology, and economics to understand and address this problem. Throughout, my methods combine both lab and field experiments, in both developed and developing countries, and I focus on establishing causal links between my variables of interest.

I ask two broad questions: first, what are the neurobiological and psychological consequences of poverty? Second, do these consequences, in turn, influence economic behavior, and could these influences perpetuate poverty? Together, these relationships could consitute a vicious cycle of poverty, in which poverty has particular psychological and neurobiological consequences, which in turn lead to behaviors that reinforce poverty.

The particular incarnation of this hypothesis that I address in this thesis is as follows: first, does poverty cause stress? Second, does stress lead to short-sighted economic decisions, which then exacerbate poverty?

In the long run, I hope that these questions will expand and crystallize into a broad area of research within cognitive science, "Psychology and Neurobiology of Poverty". The promise of this field would be to a) generate novel insights into cognitive and affective processes in an overlooked but important domain, poverty; b) in doing so, elucidate the heterogeneity of cognition and behavior across socioeconomic and cultural boundaries and beyond "normal subjects" (MIT undergraduates?); and c) inform development policy and thus alleviate poverty.

1.1 From poverty to cognition: stress and locus of control

The hypothesis that poverty leads to stress is motivated by a strong correlation between socio-economic status and baseline cortisol: in Switzerland, I find that a 1% increase in income is associated with a 3% decrease in baseline cortisol levels, even after controlling for a variety of other socioeconomic factors (Chapter 3). However, a crucial question is whether this relationship is causal. To address this question, I use two natural experiments in Kenya: in Elangata Wuas, a region populated by Maasai tribespeople, a severe drought in 2008/2009 led to the death of 46% of all livestock, which is the main income source. The proportion of livestock lost by each household is not predicted by any observable variables – in particular, rich households could not protect themselves from the loss of livestock. This makes the loss of livestock a random negative income shock to households, and thus it can be used to identify a causal effect of an increase in poverty on cortisol levels. I find that a year later, baseline cortisol is significantly higher in families who lost more livestock than others. In the second study, I use rainfall variation as an exogenous source of income variation: in Kianyaga, Kenya, a farming district populated by the Kikuyu, agriculture is the main source of income, and periods of no rain pose significant economic challenges for households. To ask if the absence of rain raises levels of stress hormones, I combine high-resolution infrared satellite imagery measuring rainfall with GPS location data for each household, and salivary samples to assay cortisol. I find that periods of no rain lead to significant increases in salivary cortisol, with a lag of 10 days (Haushofer et al., 2011b; Chapter 4). Thus, exogenous increases in poverty lead to increases in salivary cortisol, establishing a causal link between poverty and stress hormones.

In a lab experiment, I ask whether poverty has psychological consequences in a laboratory setting. I develop a laboratory paradigm for "poverty" (in a reduced sense of the word), and test to what extent this experimental manipulation affects psychological outcome variables, neurobiological markers of stress, and economic choice (Chapter 6). Subjects perform an effort task, from which they can earn income. To mimic two important aspects of poverty, different groups of subjects start the experiment with different initial endowments; in addition, after a certain number of periods, subsets of participants receive exogenous positive or negative income shocks. I then ask whether these manipulations – i.e., having less money than others, and/or having less money than previously – leads to increased levels of cortisol and self-reported stress. I find that negative income shocks lead to an increase

in present bias: subjects are more likely to prefer immediate outcomes and make time-incosistent intertemporal decisions after having suffered a large exogenous decrease of their wealth. This effect does not occur after positive income shocks. Crucially, current income is rigorously controlled, as the negative income shock group is brought to the same level of absolute wealth as the control group through the income shock; thus, income effect can be conclusively ruled out in generating the behavioral effect – it must result from endogenous preference formation.

Finally, I also study the causal effect of poverty on other psychological variables. Using a unique dataset of 60,000 households from 41 countries provided by the World Values Survey, I analyze the relationship between income and locus of control (LOC), i.e. the degree to which respondents feel that they control their lives vs. that external events determine their fate (Chapter 5). I find a strong correlation between income and LOC both within countries and across countries; this relationship is robust to controlling for a number of socioeconomic covariates. More importantly, I establish a causal link from income to LOC using instrumental variables: using the average distance of a country from icefree coasts as a natural experiment that influences countries' income, I show that income causally affects LOC when it is instrumented using distance from the coast. This result shows that poverty not only affects, but also causes differences in locus of control – an important variable both because of it affects economic productivity, and because it is closely related to mental health and depression.

In ongoing work, I ask if poverty decreases have the converse effect, i.e. led to a decrease in cortisol levels. I employ the methodology of randomzed controlled trials, pioneered in developing countries by the Jameel Poverty Actin Lab at MIT. In one experiment in Nairobi, a randomly selected sample of 300 informal metal workers receives free health insurnance for one year; a control group of 300 receives the monetary equivalent of the insurance as a cash gift, and a further control group of 300 recieves no intervention. Again I measure cortisol as an outcome variable; in this setting, however, I also obtain blood samples, which allows measuring cytokines such as the stress-related interleukin-6 in blood serum. In the second experiment, in Rarieda, Kenya, a rural district with high levels of poverty, I employ an even more direct poverty decrease: here 500 randomly chosen participants receive an unconditional cash transfer of $300 (250 to women, 250 to men), while a control group of 500 receives no transfer. Again I measure cortisol levels to assess the effect of this intervention on stress hormones. Together, these studies will show whether a causal link exists from poverty decreases to stress hormones. In a third ongoing RCT, I study the effect of community monitoring on the performance of clinics and the stress levels of health workers in Sierra Leone.

Results from these projects are expected in 2013.

1.2 From cognition to economic choice: stress and temporal discounting

The causal relationship between poverty and stress that I have begun to identify is important in its own right, as stress is a significant factor in the etiology of depression (Holsboer, 2000), and, through its impact on the immune system, a contributor to disease progression, in particular atherosclerosis (Glaser & Kiecolt-Glaser, 2005). However, it is difficult to resist asking the natural next question: does stress also affect decision-making, in ways that may exacerbate poverty?

The economic behavior I am particularly interested in is temporal discounting. Defined as the subjective devaluation of outcomes through delay, it is a pervasive feature of human behavior, and helping people overcome their bias towards the present has been shown to produce large welfare gains in developing countries (e.g. Ashraf et al., 2006). Indeed, the original observation that led me to pursue the relationship between stress and discounting was that, in both Switzerland and Kenya, poorer people not only had higher levels of cortisol, but also showed much "steeper" discounting than others. Could stress causally affect temporal discounting?

I address this question in a lab experiment in which we manipulate stress levels using the well-known Trier Social Stress Test (TSST; Chapter 7). In this task, a video-recorded mock job interview and mental arithmetic in front of a panel of judges reliably induces stress (i.e., raises cortisol and noadrenaline levels), compared to a control condition without video recording and judges. After inducing stress, we elicit discount rates using standard methods from behavioral economics. In doing so, we experimentally and econometrically distinguish between two aspects of discounting: pure time preference or impatience, captured by an exponential discount function; and present bias, captured by a quasi-hyperbolic discount function. Present bias is the more interesting of the two parameters, since it is normatively irrational: it implies time inconsistency, i.e. present-biased subjects will not follow through on (savings) plans they make today.

We find that stress strongly affects discounting, with two interesting twists: first, stress affects present bias, but not impatience; second, immediately after stress ("early" group), stressed subjects are more present-biased than controls, while 20 minutes later ("late" group), they are less present-biased than controls. Both of these effects are independently significant. In

addition, we find evidence suggesting that cortisol may drive these results: within the stress group, the magnitude of the stress-induced cortisol increase correlates across subjects with the size of the stress effect on present bias. Crucially, this correlation goes in opposite directions for the early and late groups, and is independently significant in each group.

This result is consistent with a rich literature documenting an inverted-U relationship between stress and performance, the well-known Yerkes-Dodson law (Yerkes & Dodson, 1908): in line with this hypothesis, we found that moderately increased levels of cortisol, captured in the "late" group, lead to a decrease in irrational present-biased responding, while very high levels, captured in the "early" group, have the opposite effect. However, an alternative account is that time, not levels, is the relevant factor: cortisol has both immediate and delayed effects on the brain, and it could be that the immediate effects lead to more present bias, while the delayed effects, which come to the fore when testing 20 minutes after stress, lead to less present bias. To distinguish the time and levels accounts, it is necessary to manipulate cortisol levels directly. This approach also deals with a further caveat concerning the study, namely that the correlation between present bias and cortisol we observed in our study is not conclusive proof that cortisol is causally responsible for the behavioral effects; only administration can achieve this.

I am therefore currently preparing a follow-on study in which I will administer hydrocortisone to healthy male participants, and then tests its effects on discounting behavior. Hydrocortisone is the biologically identical synthetic version of cortisol and is prescribed against rheumatoid and inflammatory diseases, allergies, and skin conditions. Crucially, to distinguish between the two hypotheses described above, hydrocortisone will be administered in two different doses: subjects will either receive 20 mg or 40 mg doses. In addition, testing will take place at two different times after hydrocortisone administration: 30 minutes, or 1 hour. Together, the combination of these conditions tests both the levels hypothesis and the time hypothesis: if cortisol levels are the crucial factor in affecting discounting, the two different doses of cortisol should affect discounting differentially, without an effect of time; conversely, if time is the crucial factor, the different test times after hydrocortisone administration should affect discounting differentially, while the different doses should have no effect.

As described above, the laboratory paradigm for "poverty" offers a further opportunity to study the relationship between poverty, stress, and discounting. In this experiment, a group of subjects receives a negative income shock, which reduces their income to the level of the "always poor" group; at this point, I elicit their discount function. These two groups have the same level of income at this stage, but they differ in whether or not they received a

negative income shock just before. I find that those subjects who received the negative income shock have significantly higher present bias than the "always poor" group, suggesting that an increase in "poverty" even in this restricted sense leads to an increase in present bias. Thus, this finding shows a direct link from poverty to temporal discounting.

In a wider context, I have also been interested in another salient feature of poverty that is closely linked to stress: violence. In a recent paper I studied the temporal dynamics of retaliation in the Israeli-Palestinian conflict (Chapter 9). We found that, contrary to previous claims, retaliation in the conflict occurs in both directions, i.e. Palestinians retaliate against previous Israeli aggression, and Israelis retaliate against prior Palestinian aggression. This paper triggered a collaboration with Israeli scientists, with whom I am currently preparing an experiment in Israel which aims to test whether behavior towards the ingroup vs. the outgroup among Israelis and Palestinians could be driven by conflict-induced stress. Israeli and Palestinian subjects will be primed with emotionally engaging information about the conflict, or with neutral primes; following these primes, we will assess a) whether they increase perceived stress and cortisol levels, and b) whether they affect behavior towards the ingroup vs. the outgroup. Specifically, using a sophisticated version of the Prisoner's Dilemma from game theory, we will be able to distinguish the effect of the prime on four motives of behavior, namely in-group trust, in-group love, out-group hate, and out-group aggression (cf. de Dreu et al., 2010, for details).

1.3 Future goals

The studies described above are the beginning of what I hope will be an in-depth research program on the psychology and neurobiology of poverty. Since this area of research is so young, there is no shortage of important follow-on questions and projects; a selection follows below:

1. My research to date has focused on stress and locus of control as psychological variables that are affected by poverty and that may in turn influence economic behavior. However, other important variables remain largely untested, e.g. optimism, self-esteem, and altruism/pro-social preferences. My future work will address whether these variables are causally affected by poverty, and whether they, in turn, have implications for economic behavior.

2. Similarly, in the neurobiological domain I have so far focused on cortisol, cytokines, and alpha-amylase (a marker of noradrenergic activity). However, other hormones and neurotransmitters have shown intriguing associations with poverty, e.g. serotonin and tesotserone (see Haushofer, 2011c, and

Eisenegger, Haushofer, Fehr, 2011, for a review). In the case of serotonin, the association seems to be additionally modulated by genetic polymorphisms. My future work will therefore study the causal relationship between poverty and serotonin and tesosterone, and their effects on economic behavior.

3. My current laboratory studies have all been run in developed countries (Switzerland, USA). With my current R01 grant, I plan to establish a behavioral testing lab in Nairobi, Kenya, that will allow to repeat the same experiments in a developing country, with subjects who have actually experienced poverty. In addition, the plan is that this lab will be accessible to cognitive scientists and behavioral economists who want to expand the populations they study to include subjects who have experienced severe poverty.

4. My research shifts into focus what looks like a serious mental health problem in developing countries: I find very high cortisol levels and staggering rates of depressive symptoms in my study populations. However, interventions to address this problem are few and far between; while simple psychotherapy interventions for developing contexts exist and show some initial promise, this research is in its infancy, and no study has rigorously tested the effects of such interventions on overall welfare and neuroendocrine markers of stress. I am currently working with Prof. David Ndetei MD, head of the Department of Psychiatry at the University of Nairobi and Director of the Africa Mental Health Foundation, to set up a randomized controlled trial that evaluates a psychotherapeutic intervention among populations in Kenya and Sierra Leone that show high rates of depressive symptoms.

1.4 Significance

The goal of my research agenda is to test whether poverty could perpetuate itself through neurobiological and psychological "vicious circles", in which poverty has particular psychological and neurobiological consequences, which in turn lead to behaviors that reinforce poverty. If that is the case, these factors could offer new possibilities for poverty alleviation in the future, and thus bring us a step closer towards solving this lingering global problem. Apart from this humanitarian purpose, I foresee scientific insights of relevance to several disciplines:

First, within cognitive and neuroscience, I hope that my research program will both provide novel insights into cognitive and affective processes in poverty, and in addition place a renewed focus on the study of cognitive and affective heterogeneity across cultures and socioeconomic strata. In development economics, my findings suggest a novel source of poverty traps, namely psychological and neurobiological channels, which can inform theo-

ries of inefficiencies and market failures in developing countries. In public policy, my findings suggest novel targets for poverty alleviation programs, namely the cognitive processes that are affected by poverty and which in turn affect decision-making. In addition, my research is relevant to public health researchers since it shifts into focus what looks like a serious mental health problem in developing countries, and suggests ways of addressing it.

Thus, my broader goal is to get psychologists and neuroscientists more interested in using their tools to address the problem of global poverty, and to get development economists and policymakers more interested in a deep understanding of behavioral and neurobiological features of poverty. My initial findings suggest that this approach holds promise, and with some luck, it may make a small contribution towards shrinking the elephant in the room.

Bibliography

[1] Ashraf, N., Karlan, D., & Yin, W. (2006). Tying Odysseus to the Mast: Evidence from a Commitment Savings Product in the Philippines. Quarterly Journal of Economics, 121, 635-672.

[2] Cornelisse, S.*, Haushofer, J.*, Fehr, E., Joels, M., & Kalenscher, T. (2011). Bi-directional Effect of Stress on Present Bias in Intertemporal Choice. Revise-and-resubmit, PNAS. (* contributed equally)

[3] De Dreu, C. K. W., Greer, L. L., Handgraaf, M. J. J., Shalvi, S., Van Kleef, G. A., Baas, K., Ten Velden, F. S., Van Dijk, E., & Feith, S. W. W. (2010). The neuropeptide oxytocin regulates parochial altruism in intergroup conflict among humans. Science, 328, 1408 –1411.

[4] Eisenegger, C., Haushofer, J., & Fehr, E. (2011). The role of testosterone in social interaction. Trends Cogn Sci, 15, 263-271.

[5] Glaser, R., & Kiecolt-Glaser, J. K. (2005). Stress-induced immune dysfunction: implications for health. Nature Reviews Immunology, 5, 243–251.

[6] Haushofer, J., & Fehr, E. (2011). The Psychology of Poverty. Working Paper, University of Zurich.

[7] Haushofer, J. (2011c). Neurobiological Poverty Traps. Working Paper, University of Zurich.

[8] Haushofer, J., Biletzki, A., & Kanwisher, N. (2010). Both Sides Retaliate in the Israeli-Palestinian Conflict. PNAS, 107, 17927-17932.

[9] Haushofer, J., Cornelisse, S., Joels, M., Kalenscher, T., & Fehr, E. (2011a). Low Income is Associated with High Baseline Levels and Low Stress Reactivity of Cortisol, but Not Alpha Amylase. Submitted.

[10] Haushofer, J., de Laat, J., Chemin, M., & Archambault, C. (2011b). Poverty Raises Levels of the Stress Hormone Cortisol: Evidence from Weather Shocks in Kenya. Working Paper, Jameel Poverty Action Lab, MIT.

[11] Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. Neuropsychopharmacology, 23, 477-501.

[12] Yerkes RM, Dodson JD (1908). The relation of strength of stimulus to rapidity of habit-formation. Journal of Comparative Neurology and Psychology, 18: 459–482.

Chapter 2

Neurobiological Poverty Traps

2.1 Summary

Poverty remains one of the most pressing problems facing the world. The advent of large-scale randomzied field experiments in development economics has opened the floor for rigorous scientific testing of poverty alleviation programs; however, the channels through which poverty perpetuates itself remain incompletely understood. In this paper, I review evidence suggesting that poverty has particular neurobiological consequences, and that these consequences in turn lead to suboptimal economic behaviors which exacerbate and perpetuate poverty. Specifically, I argue that poverty raises levels of the stress hormone cortisol, causes dysregulation and altered gene-environment interactions in serotonergic neurotransmission, and leads to increased levels of testosterone. These neurobiological effects impair executive function and exacerbate behavioral biases in economic choice, and thus contribute to the perpetuation of poverty.

2.2 Introduction

About half of the world's population live on less than 2$ a day (UN, 2007; World Bank, 2001). This lack of financial means has far-reaching consequences: 30,000 children die every day from poverty-related causes (UNICEF, 2000); in Sub-Saharan Africa, 30% of children are malnourished, and every second child never goes to school (UN, 2007); 37% of the population are illiterate, and the average African dies 28 years earlier than the average European (UNDP, 2009). Economic poverty means lack of sanitation, shelter, food, education, and healthcare; it means living in squalor, dying early, and leaving children who face similar prospects.

It's not that nobody cares. Since the 1950s, developed countries have given 2.3 trillion dollars in aid to developing countries (Easterly, 2006). However, despite this massive flow of funds, the problem persists, and some even argue that aid has contributed nothing at all to its resolution (Easterly, 2006; Moyo, 2009). Where does this leave us? Recent years have witnessed a polarization of the debate: on one side, aid enthusiasts such as Jeffrey Sachs propose addressing all problems – nutrition, education, health, etc. – in a single, concerted effort (Sachs, 2005); on the other, skeptics such as William Easterly and Dambisa Moyo argue for leaving the problem to free markets (Easterly, 2006; Moyo, 2009).

2.2.1 An experimental approach to development economics

However, over the last decade a third position has emerged: the modest, data-driven approach of randomized controlled trials (RCTs). The basic claim of this approach is that the best way to spend aid is to first find out in a rigorous, scientific manner which development interventions actually produce positive results. In practice, this takes the form of large-scale field experiments that resemble clinical trials: a randomly chosen treatment group receives an intervention such as free schoolbooks or de-worming pills, and a control group, also randomly chosen, receives no intervention (Duflo & Kremer, 2003). Due to the random selection of the treatment and control groups, any difference in outcomes between these groups can then be directly attributed to the intervention (Imbens, 2009).

For this reason RCTs have gained respect as a powerful approach to obtain true casual evidence on development interventions. Their rise to prominence was cemented by a series of significant initial successes: for instance, an early study (Miguel & Kremer, 2004) showed that the most cost-efficient way of ensuring school attendance among children in rural Kenya – orders of magnitude cheaper than more obvious candidates such as free school uniforms or meals – is treatment with de-worming pills (see also Banerjee, 2007). Another recent study found that dramatic decreases in teenage pregnancies (and by proxy, infection with sexually transmitted diseases) can be achieved by simply *telling* girls about the risks associated with unprotected sex (Dupas, 2005).

Despite these successes, the approach has its limitations: first, certain interventions, such as changes in macroeconomic policy or large-scale infrastructural projects, are difficult to randomize: e.g., one cannot randomly assign certain interest rates to the central banks of several countries. Sec-

ond, the results of field experiments might be context-specific: de-worming children might increase school attendance in Kenya, but not necessarily in other places. Finally, the most prominent criticism of RCTs is that the *channels* or mechanisms through which the interventions work may not be clear (Deaton, 2009): to name a stylized example, if de-worming increases school attendance, is this the case because the children are healthier and therefore able to attend; or because the parents don't have to spend money on de-worming pills themselves any longer and are therefore better able to pay the school fees? Note that the policy implications of the two alternative explanations are quite different: the first would argue for an expansion of the de-worming program, while the second would alternatively justify simple subsidies for school fees. It so happens that this particular example has been resolved in favor of the first explanation (Miguel & Kremer, 2004), but the point remains that the existing literature has paid somewhat more attention to project impacts themselves than to the nature of the changes that underlie them.

The first of these criticisms – not everything can be randomized – is best answered by humble acknowledgment of this limitation on the one hand, but on the other hand also by reference to the considerable successes of RCTs in areas where they are feasible (Imbens, 2009). The second point – unknown generalizability of the results of RCTs – are relatively straightforward to address in the same fashion done by older experimental sciences like biology and psychology, namely through replication in different settings. Note, however, that even replication will always leave residual doubts – how many replications in how many countries are sufficient before we recommend world-wide de-worming of children? In contrast, these lingering doubts could be much reduced by answering the third criticism stated above, namely insufficient understanding of the mechanisms driving the effects of RCTs: if we were able to identify fundamental properties of economic systems that catalyze the effects of RCTs, this would not only be an important insight in its own right, but would also turn the question about the generalizability of RCTs into one about the universality of these fundamental properties, which might be much more readily answered.

2.2.2 The question, and a caveat

The main purpose of this paper, therefore, is to work towards identifying mechanisms through which poverty might perpetuate itself, and through which poverty alleviation programs might take their effects. In particular, for reasons explained in detail below, I focus on neurobiological and behavioral channels: these have not only proven to be extremely powerful drivers of the

effects of RCTs; they are also, so far, at an extremely early stage of research, both in terms of their relation to poverty and in terms of the sophistication of the variables that have been tested, and thus hold significant promise for future research.

By necessity, some of the neuroscientific evidence I will draw on was obtained in experimental animals, such as rats and monkeys. Relating these results to the behavior of poor people is an open invitation to misunderstanding. I should therefore stress at the outset that when I draw parallels between humans and animals, they apply universally and are not limited to poor people. None of the arguments in this paper should be misconstrued as assertions that poor people are in some fashion more like other animals than richer people. Relatedly, this paper takes no stance on the nature-nurture debate of individual differences in characteristics and skills. In particular, none of the arguments that follow rely on a hereditary view of psychological and neurobiological individual characteristics; the point of this paper is not to argue that poor people are born with certain "deficits" that amplify poverty. Rather, it is to show how universal neurobiological and psychological processes can be triggered or amplified by an environment of poverty into which one *happens* to have been born. While this does not rule out hereditary mechanisms, it limits their scope. Thus, if the reader takes anything away, it should be that we might all be poor if it weren't for certain environmental and neurobiological coincidences.

2.2.3 Economic consequences of behavioral changes

A unifying feature of many of the most successful randomized trials in development economics is that they work to a large extent through behavioral channels. For instance, in an experiment in South Africa, Bertrand et al. (2005) tested the impact of a variety of psychological factors on the likelihood of bank customers to take-up a loan offer. They found that even subtle psychological manipulations had large effects on take-up; for instance, if the letter offering the loan contained a picture of an attractive young woman, male bank clients were willing to pay up to 4% more interest on the loan. (Obviously making people buy loans isn't necessarily a desirable development outcome, but it illustrates the power of such interventions.) A study in Kenya showed that households were 50 percentage points more likely to chlorinate water if the chlorination device was placed in plain sight at the source rather than kept at home (Kremer et al., 2009). As mentioned above, Dupas et al. (2005) found that teenage pregnancies among women in Western Kenya can be decreased drastically by a simple behavioral intervention: realizing that many women already become infected as school girls through

intercourse with so-called "Sugar Daddies", who in return provide financial assistance, this study simply presented schoolgirls with a short video and lesson about the risk of unprotected sex with partners of different ages, and found that this intervention resulted in a reduction in teenage pregnancies by 65% (Dupas, 2005).

Apart from these common-sense behavioral interventions, a small group of RCTs has begun to use theory and evidence from behavioral economics and psychology to develop and improve development interventions. In particular, one prominent group of RCTs takes advantage of a particularly well-understood feature of human decision-making to improve economic outcomes: it is a well-established fact in behavioral economics and psychology that people are hyperbolic temporal discounters. This term refers to the finding that, as outcomes are delayed into the future, their subjective value decreases hyperbolically (i.e. proportional to delay). This function implies that decision-makers are time inconsistent: they will procrastinate and neglect to execute the savings plans they make today; they overspend and undersave relative to their own long-run preferences, give in to temptation at the expense of achieving their long-term goals (Laibson, 1997; Mazur, 1987; Mischel et al., 1989).

This universal feature of human decision-making suggests ways in which desirable economic outcomes can be achieved. For instance, Thaler & Benartzi (2007) took advantage of the hyperbolic shape of the discount function to increase retirement savings: they offered participants to commit *future* salary increases to their savings plan. In doing so, they subjected the subjective cost of not immediately consuming the salary increase to temporal discounting, thus making it less dramatically felt by respondents. As a result, retirement savings increased substantially (Thaler & Benartzi, 2007).

In the development economics literature, a small number of RCTs have utilized this feature of human decision-making as a vehicle to improve welfare. Duflo et al. (2009) took advantage to hyperbolic discounting to address the problem of insufficient fertilizer use in developing countries: many farmers want to use fertilizer, but they postpone buying it until the beginning of the season, when financial liquidity is low. Duflo et al. counteracted this tendency to procrastinate by offering a small discount on fertilizer purchases to farmers at the time of harvest, i.e. coinciding with greatest financial liquidity. This small intervention significantly increased fertilizer use and thus raised income and welfare.

Similarly, Ashraf et al. (2006) used the evidence on hyperbolic discounting to develop a way to help consumers in developing countries to increase their savings rates. Economic theory predicts that sophisticated time-inconsistent individuals should value commitment devices, i.e. mecha-

nisms that bind them to a particular savings plan once they have made it (Laibson, 1997). Ashraf et al. (2006) offered bank customers the Philippines a simple commitment savings device – essentially, simply a voluntary lock on the account, similar to a piggy bank, with no other benefits. The authors found not only that the commitment device was very popular among bank clients, but it also led to 84% increases in household savings rates. This result suggests, first, that many individuals save less than they themselves would like, and that therefore there is scope for behavioral "nudges" which bring behavior closer to the level desired by the individual "long-run self"; and second, that these nudges can be very effective.

Together, these examples illustrate three points. First, development interventions at the level of individual persons or households can have significant positive economic consequences. Second, interventions that operate through behavioral changes are among the most cost-effective and successful of these interventions. It is important to stress here that none of these interventions trick or mislead participants in any way; they simply take advantage of psychological regularities that we all share, and offer people an opportunity to bring their own behavior closer to what they themselves desire. Third, note that many of the behavioral interventions mentioned above – even though they are shining examples of the power of behavioral changes in alleviating poverty – are based on everyday, folk psychology: it doesn't take a cognitive scientist to come up with the hypothesis that pictures of pretty women might inspire customers to purchase a product. On the other hand, the few RCTs that did take advantage of the rich body of knowledge in behavioral economics and psychology, such as those employing the evidence on hyperbolic discounting mentioned above, opened up powerful and previously unexplored mechanisms for improving welfare in developing countries.

2.2.4 Behavioral characteristics of poverty

Thus, small behavioral changes can have substantial economic consequences at the household level, leading to better use of limited resources, increased savings, and improved health. Two further questions arise from these insights. First, what could be achieved through interventions that take even fuller advantage of the rich toolkit and existing knowledge of neurobiology, experimental psychology, and behavioral economics? Second, what could the starting point for developing such interventions be? In my view, the most obvious candidates for behaviors and psychological variables which might affect poverty are those which themselves are specific to, and potentially results of, poor contexts. Thus, the most convincing answer to this question is a further question: What are the behavioral characteristics of poverty?

In the following, I briefly summarize the behavioral consequences of poverty in two domains: psychological outcomes and economic choice; in addition, I briefly consider the effect of these domains on one another.

2.2.4.1 Psychological characteristics of poverty

Several lines of research suggest that specific behaviors and psychological states may be particularly pronounced in settings of poverty (Bertrand et al., 2005).

First, a well-known literature has investigated the relationship between self-reported happiness and income both within and across countries. Initially it was found that happiness correlated with income within countries, but not across countries (Easterlin, 1974); thus, surprisingly, the poorest person in a developed country such as the United States was on average just as satisfied with her life as the poorest person in a developing country like Kenya, even though the differences in absolute income between the two were enormous. However, this finding was recently overturned: using newer and better data, Hagerty & Veenhoven (2003) and Stevenson & Wolfers (2008) showed that there was a strong correlation between self-reported happiness and income not only within countries, but also across countries: people in richer countries were, on average, happier than those in poor countries.

On the other end of the happiness spectrum, a related literature has investigated the prevalence of clinical depression in developed countries. The most striking finding of these studies is that the prevalence of depression in developing countries is much higher than in developed countries: while the point prevalence figures are around 5-10% in Europe and North America, developing countries report numbers such as 19% (Lebanon, Mexico), 20% (Thailand), 24% (Uganda), 39% (Dominican Republic) and 40% (Cuba; Bolton et al., 2004; Garcia-Alvarez, 1986; Thavichachart et al., 2001; Sobocki et al., 2006; Patel et al., 2003). Within countries, low-income population groups show prevalence rates that are 1.5 to 2 times higher than those of high-income groups (WHO, 2001). Depression is projected to be the leading cause of disease burden worldwide by 2030 (WHO, 2001), and is already the leading cause of loss of disability-adjusted life years (DALYs) in Sub-Saharan Africa, ahead of malnutrition and cancer (World Bank, 1993).

A central element in the etiology of clinical depression is stress, and the associated stress hormone cortisol: 80% of all patients with depression have histories of chronic stress or stressful life events (Hammen, 2005), and depression is marked by dysregulation of the stress hormone cortisol (Holsboer, 2000). Another major biological player in depression is serotonin; depressed patients show serotonergic dysregulation, and selective serotonin reuptake

inhibitors (SSRIs) are among the most effective drugs for the treatment of depression (Schildkraut, 1965; Fournier et al., 2010). Putting these facts together with the evidence on high prevalence of depression in developing countries suggests that poor people might exhibit higher levels of cortisol and dysregulated serotonergic functioning; this relationship is a main focus of the remainder of this paper.

Evidence on the relationship between other psychological variables and poverty is scarce. The most robust association with poverty, apart from stress and depression, is that of self-esteem: individuals with high SES consistently show higher self-esteem that those with lower SES (for a review, see Twenge & Campbell, 2002). In cognitive neuroscience, Martha Farah and her colleagues have provided evidence showing a correlation between poverty and components of cognitive functioning; they find that children from middle socioeconomic status (SES) families substantially outperform those from poorer families on tests of working memory, language ability, and executive function (Noble et al., 2005, 2007). Evans & Schamberg (2009) confirmed that childhood poverty was associated with impaired working memory performance in young adults, and additionally indicated that this relationship might be mediated by chronic stress. A smaller number of papers suggests a correlation between poverty and external locus of control, i.e. the perception that one's life outcomes are determined mostly by external events rather than one's own behavior (Maqsud & Rouhani, 1991; Sherman & Hofmann, 1978). Finally, a correlation between poverty and optimism has been suggested: poor people tend to view their future more negatively than richer individuals (Scheier & Carver, 1985; Robb et al., 2009; Lynch et al., 1997; Taylor & Seeman, 1999).

However, conclusive evidence on the nature of the relationship between poverty and these variables is lacking: almost all studies were conducted in developed countries, and most damagingly, demonstrations of causality are missing. However, in an ingenious field experiment, Spears (2010) has begun to address these problems and attempted to establish a causal relationship between poverty and psychological outcomes in a developing country. He randomly assigned poor participants in India to one of four conditions: they could either be rich or poor, in the sense that they received either two (rich) or one (poor) good from a choice set of three options. In addition, each participant could either be in a "choice" or a "no choice" condition, where the former meant that participants could choose which item(s) from the choice set they wanted to receive, whereas in the latter case the items were randomly assigned. Spears then asked participants to perform two tasks that are frequently used to measure cognitive control: squeezing two handlebars for as long as possible, and performing a Stroop-like task. In this task, partic-

ipants have to name the number of items in a display, which, confusingly, are numbers themselves; thus, a display might be "3 3", in which case the correct answer would be "two" since there are two items in the display. Spears found that participants performed worse on the two cognitive-control tasks if they had been randomly assigned to both the poor and the choice conditions. Thus, requiring people to make a choice appeared to deplete cognitive control, but this was only true for poor participants.

2.2.4.2 Economic-choice characteristics of poverty

In addition, an emerging economics literature suggests that poverty may be associated with particular patterns of economic choice, in particular with regard to time preference and risk aversion: poor individuals tend to be more impatient and more risk-averse than richer individuals.

In an early paper, Lawrence (1991) estimated Euler equations from US panel data and found a negative correlation between time preferences and income. She concluded that savings behavior may be affected by differences in discounting and hence may contribute to the observed heterogeneity across socioeconomic groups.

In a more recent paper, Tanaka et al. (2010) report a field experiment in Vietnam which studies the relation between time preferences, risk preferences, and wealth. Subjects were presented a number of payment options and were asked to choose between a smaller amount they could receive the same day, or a larger amount to be paid in the near future; alternatively, they chose between a small, low-risk amount vs. a larger, riskier amount. The authors found that poorer people discount future payoffs at a higher rate and are hence less patient; in addition, they are more risk-averse than richer individuals. This pattern should also affect savings behavior and wealth accumulation over time, suggesting that initial poverty could be obstructive to further economic development. Note that it remains somewhat unclear in these studies whether present-biased time preferences and risk aversion might be explained by liquidity constraints. More importantly, the important question whether the relationship between poverty and economic choice is causal remains unanswered by these studies.

2.2.4.3 Economic-choice consequences of psychological variables

Finally, a small literature suggests that changes in psychological variables may have economic consequences. On a general level, the macroeconomic effects of depressive disorders are well-known; they include unemployment, absences, and at-work performance deficits (for reviews, see Lerner & Henke,

2008; Crisp, 2007). In fact, impairment in occupational functioning is among the diagnosis criteria for depressive episodes (American Psychiatric Association, 1994), and it is estimated that the economic cost of depression in Europe amounts to around 1% of Europe's GDP (Sobocki, 2006). In addition, a small number of studies in experimental economics suggests that experimentally induced happiness and related emotions lead to improved economic performance, such as higher effort (Oswald et al., 2009), productivity (Argyle, 1989, 2001), and creativity (Amabile et al., 2005).

Thus, poverty appears to be associated with particular psychological and behavioral outcomes. In the following, I outline three neurobiological mechanisms which might underlie these effects. Specifically, in Sections 2, 4, and 6, I show that poverty raises levels of the stress hormone cortisol, causes dysregulation and altered gene-environment interactions in serotonergic neurotransmission, and raises levels of the sex hormone testosterone. These neurobiological effects in turn have behavioral consequences; specifically, they impair executive function and exacerbate behavioral biases in economic choice. These consequences are discussed in Sections 3, 5, and 7. Section 8 addresses some emerging questions, and Section 9 concludes the paper.

2.3 Cortisol and Poverty

2.3.1 Cortisol Basics

Cortisol is the body's major stress hormone. It is synthesized by the hypothalamic-pituitary-adrenal (HPA) axis: in response to external stressors, the hypothalamus in the midbrain secretes corticotrophin-releasing hormone (CRH), which in turn controls the release of adrenocorticotropic hormone (ACTH) from the pituitary gland; ACTH then causes the release of cortisol from the adrenal gland. Two factors give cortisol its prominent role in stress: first, it is released in response to both psychological and physiological strain on the organism. In the physical domain, it increases following bodily injuries, physical exertion, illness, and extreme temperatures. In the psychological domain, cortisol increases in response to social stressors such as having to give a speech in front of a panel of judges, performing mental arithmetic, or enduring physically unpleasant situations like immersion of one's hand in cold water (Kirschbaum et al., 1993; Ferracuti et al., 1994).

Second, cortisol in turn has exactly those effects on the body that one would expect from a stress hormone; in particular, it increases blood sugar to levels that prepare the organism to deal with stress. Moreoever, cortisol exerts a direct and broadly suppressive effect on the immune system;

in particular, it suppresses pro-inflammatory cytokines such as interleukin 6 and interleukin 1 (Straub, 2004; Wilckens, 1995). However, chronic elevations of cortisol appear to have the opposite effect, leading to permanent mild elevations of cytokine levels (Kiecolt-Glaser et al., 2003). These cytokine elevations then contribute directly to disease onset and progression, e.g. in atherosclerosis and cancer (Steptoe et al., 2001, 2002; Aggarwal et al., 2006; Coussens et al., 2002; Ross, 1999). Thus, while transient cortisol elevations are adaptive and protective, permanently high cortisol is physiologically damaging, quite apart from the psychological effects.

2.3.2 Cortisol and Poverty

Does cortisol relate to socio-economic status or poverty? An increasing number of studies suggest that this is indeed the case.

2.3.2.1 Studies in children

A sizeable literature investigates the effect of family socioeconomic background on the cortisol levels and cognitive outcomes of children. In these studies, socioeconomic status is usally defined as a compound measure of parental education and income.

Evans & English (2002) collected overnight urine samples from a sample of 287 rural white children who were either from low-income or middle-income families. The urine samples were assayed for cortisol, epinephrine, and norepinephrine, all markers of stress. Evans & English found that children from poor families had significantly higher levels of overnight cortisol and epinephrine than those from richer families, while levels of norepinephrine were not significantly different across these groups.

In a later study also by Gary Evans, 207 children from the same sample, now aged 13, were again tested for overnight cortisol, and this measure was related to the duration during which each participant had experienced poverty during childhood (Evans & Kim, 2009). There was a robust positive association between the two, with children having spent more time in poverty exhibiting higher cortisol levels than others. In contrast, cortisol levels did not correlate with concurrent poverty levels.

Using a longitudinal approach, Chen and colleagues (2010) studied 50 children from families of varying socioeconomic background. Notably, they collected saliva samples over a period of 2 years, with 4 daily samples, repeated on two consecutive days on each occasion. Measurements were taken in 6-month intervals. The authors used area under the curve (AUC) of the diurnal profile as the cortisol measure of interest, and focused on family sav-

ings as an indicator of socioeconomic status. They found that while there was no initial relationship between savings and cortisol, the *change* in cortisol AUC over the course of two years was predicted by SES, with children from low-SES families showing a steeper increase in cortisol AUC than children from families with higher SES.

Lupien et al. (2000) measured cortisol level in a sample of 217 children aged 6-10, and related them to the socioeconomic status of the children's families. High-SES children had significantly lower cortisol levels than medium- and low-SES children across all age groups. The difference between medium-SES and low-SES children was significant only for the 10-year old group. The authors suggest that maternal depression may be a mediator of this finding, as the mothers' score on the depressive subscale of the Derogatis stress profile was correlated with the child's cortisol level.

How long does the influence of parental SES on children's stress levels last? In a follow-up study to the one discussed above, Lupien et al. (2001) again measured cortisol levels in 302 school-age children, this time in the age range from 6-16. They replicated their previous finding of higher cortisol levels in low-SES children in the age range from 6-10, but found that no such relationship obtained for older children, i.e. after the transition to high-school the relationship between SES and cortisol disappeared. One possible explanation of this finding is that the influence of parents and their socioeconomic background on children's stress levels attenuates as children get older and high school peers assume a more central role.

An important source of concern regarding SES-cortisol correlations is the direction of causality: while it is plausible that low SES causes stress and raises cortisol levels, it may also be the case that high cortisol levels lead to bad health, which in turn brings down income, leading to a pathway from cortisol to SES rather than vice-versa. One advantage of studies in children is that reverse causality is a more difficult proposition, i.e. it is harder to argue that children have low SES because of their cortisol levels. One study capitalized on this argument by studying extremely young children: Saridjan et al. (2009) measured salivary cortisol throughout the day in 366 infants aged between 12-20 months. They found that both the AUC and the Cortisol Awakening Response (CAR) were larger in infants whose families had low incomes compared to infants from high-income families. For children at this extremely young age, it is unlikely that family SES is low because of the children's cortisol levels; rather, the causality is more likely to run in the other direction. Nevertheless, genetic and very early developmental factors cannot be ruled out; indeed, estimates for the heritability of cortisol levels are around 60% (Bartels et al., 2003). Studies establishing causality are therefore sorely needed.

In addition to these correlational studies, one study has provided evidence for a cortisol-related gene-environment interaction. Ouellet-Morin et al. (2009) measured the genetic contribution to morning cortisol levels, either right at awakening or some time later in the laboratory, in 6-month old twins. The authors found a significant gene-environment interaction, in that monozygotic twins showed a markedly higher correlation in their cortisol levels compared to dizygotic twins in the laboratory sample, but only when family adversity was high (i.e. SES was low). This finding is in line with the so-called "diathesis-stress" hypothesis, which states that an individual's genetically determined vulnerability to stress comes to bear only in resource-poor settings. Similar gene-environment interactions have been observed for a polymorphism of the serotonin transporter gene; these will be discussed below.

These findings strongly suggest an association between parental SES and children's cortisol levels. Nevertheless, a small number of other studies are more equivocal or even point in the other direction: Cutuli and colleagues measured salivary cortisol in 66 homeless children aged between 4 and 7, both before and during a set of cognitive tasks, such as a reasoning task, a Stroop task, and a vocabulary test. They used a measure of "socioeconomic risk", which was a variable composed of indicators for neighborhood quality, parental education, parental unemployment, lack of family income, and ability of parents to pay rent, and compared this measure to children's cortisol levels throughout the task. No association between either morning cortisol or task-related cortisol increase and socioeconomic risk was found. However, a graded measure of income was absent from the socioeconomic risk measure, and its six elements were simply present-absent dichotomies; this simplification is likely to have led to some loss of information. In addition, it may be the case that socioeconomic status is not reflected in the cortisol increase in response to cognitive tasks.

Kraft & Luecken (2009) measured cortisol levels in 94 young adults both before and after they performed a speech task. Throughout the task, and prior to it, children from families where the parents had divorced showed *lower* levels of cortisol and children from intact families. A similar pattern was observed for family income, i.e. children from richer families had higher cortisol levels than children from poorer families. It is unclear what gives rise to this finding.

Finally, Dulin-Keita et al. related the cortisol levels of 148 children with an average age of roughly 8 years to the quality of the neighborhoods in which the children grew up. Neighborhood quality was measured through "disorder" indices. The authors found that in African-American children, lower neighborhood quality was associated with significantly *lower* serum

cortisol levels, while this relationship was reversed, although not significant, in Caucasian children. The authors suggest that getting used to and learning to cope with an environment of disadvantage may lower cortisol levels in the long run.

Despite these contradictory findings, the majority of studies do find a negative association between parental SES and children's cortisol levels. From this result, naturally the question arises to what extent the adverse developmental outcomes that are characteristic of an environment of poverty might be due to the stress channel. In one of the first studies to relate socioeconomic status, levels of stress hormones, and cognitive outcomes, Evans & Schamberg (2009) found that working memory performance in young adults was lower for individuals coming from poor families; moreover, the coefficient on poverty became non-significant when the authors controlled for allostatic load during childhood. Allostatic load was a composite measure which included overnight urine levels of cortisol, epinephrine, and norepinephrine, body mass index, and resting blood pressure. This finding suggests that a significant proportion of the relationship between poverty and adverse cognitive outcomes can be explained by chronic stress during childhood.

2.3.2.2 Studies in adults

Does the association between SES and cortisol levels that is obversed in children also hold in adults?

In an early study, Arnetz et al. (1991) measured cortisol levels in a sample of 354 Swedish blue-collar workers before and after a subset of these workers lost their jobs. The authors measured serum cortisol levels along with a number of other health indicators, and found that cortisol levels were significantly higher in those workers who lost their jobs.

Cohen et al. (2006a), in one of the largest studies up to that point, obtained daily cortisol profiles from 781 middle-aged adults who were participants in the CARDIA study. The authors found that lower income and education were associated with higher evening cortisol levels. This finding translated into a larger AUC for poor than for richer participants, and a flatter decreasing slope in the cortisol profile throughout the day. This study is noteworthy for its size and the relatively involved cortisol sampling technique, with 6 daily samples per respondents. The fact that the results clearly support a relationship between cortisol and SES suggests that studies which did not find this relationship may not have large enough sample sizes or a sufficient number of measurement timepoints.

In another study by Sheldon Cohen, he and coauthors tested 193 adults of various ages for cortsiol and catecholamines (Cohen et al., 2006b). Specif-

ically, their subjects provided seven daily salivary cortisol samples over three days, and 24-hour urine samples for catecholamines over 2 days. Cohen et al. found that persons with low SES, as measured by income and education, had higher overall levels of cortisol. Similarly, they had higher epinephrine levels, while a marginal effect was found for higher levels of norepinephrine.

In yet a larger study, Li et al. (2006) obtained two daily cortisol measures – one in the morning, one in the evening – from a sample of 6335 45-year-olds who were members of the 1958 British Birth Cohort Study. In addition, the authors measured lifetime socioeconomic position by categorizing the occupations of the respondents themselves and their fathers into high- and low-class jobs. There was a robust relationship between socioeconomic position (SEP) thus defined and "extreme" values on the morning cortisol sample, as well as between SEP and cortisol AUC, and between SEP and an "abnormal" diurnal cortisol decline (where abnormal was defined as having a morning cortisol value$\leq$ 7.5 nmol/L, or an evening cortisol value that is more than 80% of the morning value). Poor individuals showed higher overall levels, higher AUC, and more abnormal diurnal patterns.

This evidence suggests that high levels of stress and its physiological markers cortisol may be consequences of poverty. Note, however, these studies were conducted in developed countries; it remains unclear whether a similar relationship exists in developing countries. More importantly, these findings are merely correlational and therefore do not justify conclusions about whether poverty causes stress or vice-versa.

Two very recent studies have attempted to resolve this correlation-causation dilemma, and measured the impacts of development programs on stress levels: Fernald & Gunnar (2009) measured cortisol levels in children who had been exposed to the Mexican PROGRESA program – a comprehensive conditional cash transfer program with a focus on health and education. The authors found that children who had been exposed to the program exhibited lower baseline cortisol levels than those children who had not been in the program. In another study, Fernald et al. (2008) investigated responses to stress and depression questionnaires in a sample of South-African respondents after they were randomly assigned to receive a loan. Those who had received loans showed lower levels of depressive symptoms than the control group; interestingly, however, questionnaire-assessed stress levels were higher after receiving a loan than in the control group, possibly due to the stress induced by having to pay back the loan at a high interest rate (200% p.a.). Even though the former study was in children rather than adults, and the latter cannot unambigulously be called a reduction in poverty since people had to pay large interest on their loans, these findings lend weight to the hypothesis that poverty (reductions) may have psychological consequences.

2.4 Cortisol and Economic Choice

The previous section summarized findings suggesting that poverty causes increased levles of cortisol. This section reviews evidence that this increase in cortisol levels, in turn, has cognitive and behavioral consequences.

There is a vast literature on the cognitive effects of cortisol (for reviews, see McEwen & Seeman, 1999; McEwen, 2004; McEwen & Sapolsky, 1995; de Kloet et al., 1999; Lupien et al., 2007, 2009; Kim & Haller, 2007). Much of this research has revolved around memory. This is due to the fact that corticosteroid receptors are most prominently expressed in the hippocampus, which is a crucial structure for spatial and declarative memory, and in the prefrontal cortex, which serves important working memory functions (de Kloet et al., 1999). Generally, moderate levels of stress and cortisol are thought to be performance-improving; however, severe and prolonged exposures lead to performance decrements (McEwen & Seeman, 1999; McEwen, 2004).

Since the effects of cortisol on memory have been discussed extensively elsewhere (McEwen & Seeman, 1999), I focus here on its effects on decision-making and economic choice. Klein (1996) reviews the evidence of the effects of actue stress from the military and human factors literature human decision-making. He argues that the main cognitive consequences of stress for cognition are narrowed attention and focus on salient cues, as well as reduced working-memory capacity. The consequences of these effects are decision-making strategies that are simpler than usual, a decreased reliance on analytical in favor of "recognitional" or salience-based strategies, and less complete mental simulations.

An early study on the effects of stress on decision-making by Keinan (1987) offers support for this argument. Keinan put participants in a situation where they expected either controllable or uncontrollable electric shocks while performing a decision-making task. In the task, subjects had to choose the correct verbal analogy from a set of six options. In the uncontrollable threat condition, they were told in addition that every now and then throughout the experiment, they would receive mild electric shocks to their non-dominant hand. The controllable condition was identical, with the exception that subjects were informed that the shocks were contingent on their performance on the task. In the control condition, subjects were not told that they would be shocked during the experiment. In reality, no shocks were administered in any of the conditions; any effects on performance were thus due to expectation rather than experience. The author found that subjects performed significantly worse on the task when they expected to be shocked: specifically, they got fewer answers correct, scanned the alternatives in non-

systematic ways, and stopped scanning alternatives earlier when they expected a shock than when they did not. No differences were found between the controllable and uncontrollable conditions.

In one of the first experiments testing the effect of stress on economic choice, Gray (1999) used the temporally extended choice task by Herrnstein et al. (1986) to study the effect of stressful emotional context on decision-making in this task. Subjects are asked to advance slides on a projector, using one of two buttons. They earn money for displaying the maximum number of pictures in a given time, i.e. advancing the projector as quickly as possible. The speed at which the projector can be advanced is determined by a central fixation spot, which on every trial takes a set number of seconds to disappear; subjects can only advance the projector once the fixation point has disappeared. Crucially, subjects can influence the fixation duration on each trial by choosing the button which they press to advance the projector: one button, the "bad" button, speeds up the immediately consecutive trial, but slows down the four that follow, resulting in an overall temporal cost. In contrast, the "good" button delays the immediately following trial, but speeds up the four that follow, resulting in an overall temporal gain. Thus, this task can be compared to an Iowa Gambling Task over time, with subjects having to keep track of the temporal (rather than probabilistic) consequences of each button press; a preference for the "bad" key can be interpreted as short-sighted intertemporal choice.

Gray established a stressful vs. non-stressful context for this task by presenting the fixation spot on a background of either neutral or negative emotional pictures from a standard picture database. He found that subjects who were exposed to stressful, negative pictures performed worse on the task, i.e. exhibited a larger preference for the "bad" key, and as a result earned less money than the control group. Thus, it appears that inducing a stressful emotional context leads to a performance decrement on a concurrently performed decision-making task.

Preston and colleagues (2007) tested subjects on the well-known Iowa Gambling Task (Bechara et al., 1994, 1999). In this task, subjects are presented with four decks of cards, A, B, C, and D. Subjects draw cards from decks of their choice and gain or lose the number of points shown on each card they draw. Decks A and C are "disadvantageous" in that they lead to long-run losses, even though the initial cards have high gains. In contrast, decks B and D are "advantageous" in that they lead to long-run gains, despite high initial losses. Over time, normal subjects learn to choose from the advantageous decks; inability to learn this behavior can be interpreted as suboptimal economic decision-making. Preston and colleagues found that subjects who were anticipating to give a public speech while they performed

the Iowa Gambling Task were significantly slower at learning the optimal response pattern, resulting in lower overall gains. In addition, the effect was differentiated across men and women, with a performance decrement under stress for men, and a performance improvement for women. The authors argue that given the somewhat better average performance usually observed for men on this task, this finding is consistent with an inverse U-shaped relationship between stress and task performance. An alternative possibility, and a very common problem in this literature, is the hormonal fluctuations make it more difficult to obtain clean data from female subjects.

An obvious explanation for this finding not related to stress, which the authors acknowledge, is that the impending speech occupied working memory resources; this is a somewhat distinct explanation from a simple effect on stress, which should also operate without working memory impediment.

This criticism applies to a lesser extent, however, to another study which assessed behavior on the Iowa Gambling Task under conditions of stress; van den Bos et al. (2009) tested subjects on the IGT, but after they had been exposed to a social stressor. This task, the well-known Trier Social Stress Test, requires subjects to give a speech in front of an interview panel, and perform a challenging arithmetic task. It has been shown that it reliably increases stress and cortisol levels (Kirschbaum et al., 1993; Kirschbaum, 1999). Van den Bos and colleagues found, again, that male subjects performed significantly worse at the IGT after they had been exposed to stress, and as a consequence earned less money in the experiment. Again findings were inconclusive for women, presumably due to the issues mentioned above.

Other tasks control for this confound. Porcelli & Delgado (2009) used the well-known cold-pressor task to study the effects of stress on financial decision-making. In this task, subjects are simply asked to hold their hand in ice-cold water for 2 minutes. The control group hold their hand into room-temperature water. Note that this task does not suffer from the working memory confounds described above for subjects facing the prospect of giving a speech.

The authors then presented subjects with a financial risk-taking task, in which they could choose between two lotteries; for instance, they might be asked whether they would rather win \$0.25 with 80% certainty, or win \$3 with 20% probability. Risk-taking was defined as choosing the low-probability, high-return option. Similar trials were presented in the loss domain; here, participants might be asked, for instance, whether they would prefer losing \$0.25 with 80% probability, or losing \$3 with 20% probability.

The standard finding in risk preference tasks like this one is what has been termed the reflection effect: subjects are risk-averse in the gain domain, i.e. when the expected values of the gambles are equal, they prefer

the gamble with the higher probability; in contrast, they are risk-seeking in the loss domain, i.e. prefer smaller probabilities of larger losses to greater probabilities of small losses. This behavior is often seen as an anomaly in economics, since paying a risk premium is not consistent with behavior that maximizes expected value.

Porcelli & Delgado found that the cold pressor task reliably induced stress in that it raised subjects' skin conductance response, and subjects' recognition memory for a list of previously studied words was impaired, as is expected for memory performance after stress. Most importantly, however, the authors also observed an effect on financial risk-taking: subjects in the stress group showed an enhanced reflection effect compared to those in the control group, i.e. they were more risk-averse in the gain domain, and more risk-seeking in the loss domain. Thus, they exhibited an increase in non-normative, non-profit maximizing economic behavior under stress.

In addition to these effects on financial decision-making, stress induced by the Trier Social Stress Test has also been suggested to affect social cognition: Takahashi et al. (2005) exposed subjects to the TSST and then tested them on a questionnaire called the General Trust Scale, which measures interpersonal trust. Subjecst who had higher cortisol responses to the TSST showed lower trust scores on this questionnaire, suggesting that stress may make them less trusting in economic games. However, Takahashi did not test their behavior in actual economic exchanges, leaving this question unanswered and fodder for future research.

Most of the studies cited above took place in the laboratory. To overcome the concerns about external validity associated with this setting, Coates & Herbert (2008) studied traders on in a real-life setting on a London trading floor. The authors observed 17 traders for 8 consecutive business days, taking two saliva samples a day, at 11am and 4pm. They found that traders had higher cortisol levels, and higher variance in these levels, if their trading volatility was high, i.e. when their profits and losses (P&L) had high variance. In addition, the authors used option prices as a measure of anticipated market volatility; this approach is useful because options indicate to what extent traders value the ability to buy an asset at a fixed price in the future, thereby hedging against uncertainty. Indeed, the authors found a strong correlation between average cortisol levels and implied volatility as measured by option prices. It remains unclear in which direction causality runs; however, the finding further associates cortisol with particular behaviors, and lends further weight to the hypothesis that altered cortisol levels may have has economic consequences.

2.5 Serotonin and Poverty

In this section, I review evidence showing that poor people exhibit differential serotonergic function compared to richer people. In particular, socio-economic status, as measured by income and education, has been shown to be reliably associated with both serotonergic responsivity, and with a particular polymorphism of the serotonin transporter gene.

2.5.1 Serotonin Basics

Serontonin is one of the major neurotransmitters in the central nervous system of humans and other animals. It is synthesized from tryptophan, which is ingested with the diet, and released in the CNS by serotonergic neurons located in the raphe nuclei of the brainstem. These neurons project widely to both the neocortex and subcortical structures, including areas that are important for emotion regulation and reward processing, such as amygdala and basal ganglia (Berger et al., 2009). Serotonin is thought to be a major player in the etiology of depression; patients suffering from depression exhibit lower levels of serotonin (Schildkraut, 1965), and prolonging the presence of serotonin in the synaptic cleft with selective serotonin reuptake inhibitors (SSRIs) is a well-known and effective treatment for depression (Fournier et al., 2010).

There is notorious and profound disagreement in the literature about the precise function of serotonin. What is clear is that serotonin has far-reaching effects on mental health, and a plethora of accounts accord it an important function in anxiety (Lesch et al., 1996; Wise et al., 1972), depression (Deakin & Graeff, 1991), stress reactivity (Greenberg, 2000), as well as impulsivity, behavioral inhibition, and aggression (Manuck et al., 1998; Gray, 1982; Soubrié, 1986). While all of these disorders and behavioral patterns are associated with dysregulation of serotonergic neurotransmission, a unifying account remains elusive (Robbins & Crockett, 2010; Dayan & Huys, 2009). Regardless, it will emerge in the following discussion that serotonin has been credibly linked to poverty.

2.5.2 Serotonin and Poverty

In the first study in this vein, Matthews and colleagues (2000) studied the relationship between socio-economic status (measured by income and education) of 139 adult men and women to their serotonergic responsivity in the fenfluramine challenge. In this protocol, fenfluramine is administered and induces serotonin release and reuptake inhibition. This increase in serotonin

availability causes the release of the hormone prolactin by the pituitary gland into the blood stream; thus, the degree of serotonergic responsivity can be measured by prolactin in blood serum (Quattrone et al., 1983; Yatham & Steiner, 1993). The authors found that low-SES individuals exhibited lower PRL increases in the fenfluramine challenge than high-SES individuals. In other words, low SES was associated with blunted serotonergic responsivity. This effect was mainly due to income, as it reached significance considering only income or income and education together, but not education by itself. Differences in impulsivity in low-SES individuals appear not to account for the relationship. The authors speculate that low serotonergic responsivity in low-SES individuals could be explained by higher exposure to stressful environments in this population. This hypothesis is consistent with studies showing that monkeys who are deprived of their mothers early in life show lower levels of the serotonin metabolite 5-HIAA in their cerebrospinal fluid than those who are not deprived (Higley et al., 1992), suggesting lower serotonergic responsivity in these animals.

Interestingly, in monkeys this relationship between maternal deprivation is mediated by a length variation in the 5-HTTLPR gene: in monkeys who experienced maternal deprivation, those animals who were homozygous for the long allele of this gene had with higher 5-HIAA concentrations than heterozygous animals, while this difference by genotype was not seen in monkeys who had not experienced maternal deprivation (Bennett et al., 2002).

This finding was confirmed by a similar study in humans, in which Manuck and colleagues (2004) related the socio-economic status (measured by income and education) of 139 adult men and women to their serotonergic responsivity in the fenfluramine challenge. The authors found that carriers of at least one 5-HTTLPR short allele (SS, L/S) showed a relationship between SES and PRL increases during the fenfluramine challenge, with lower-SES individuals exhibiting lower serotonergic responsivity. In contrast, individuals who were homozygous for the L allele showed no such relationship.

This finding was somewhat qualified by another study by Manuck and colleagues (2005), conducted at the community level: these authors obtained a measure for the socioeconomic status not of individuals, but of entire communities in the United States, which consisted of median income, percentage of households below the poverty line, unemployment, home ownership, ratio of rental price to income, and education. The authors found that individuals who lived in low-SES communities had a decreased PRL response to the fenfluramine challenge than persons in high-income neighborhoods. In contrast to their previous study showing that genetic variation in the 5-HTTLPR gene modulated the relationship between SES and response to the fenfluramine challenge, this modulation was not observed at the community level.

Williams et al. (2008) studied the effect of 5-HTTLPR genotype on stress reactivity in a social stress task similar to the Trier Social Stress Test (Kirschbaum et al., 1993). Participants had to recall an event that angered or saddened them, in front of a panel of listeners; the control task consisted of reading a neutral text. Settings of this type have been shown to reliably induce stress in participants. The authors found that low-SES individuals had higher responses to the social stressor. In addition, the 5-HTTLPR genotype affected stress reactivity in this task: participants with two L alleles showed higher responses in terms of diastolic and systolic blood pressure than L/S and S/S individuals. However, SES and genotype did not interact; the effect of SES on stress reactivity was of similar magnitude for both S/S and L/L individuals. It is surprising that the L genotype was associated with higher stress reactivity, since the S allele is normally considered to be the vulnerable one.

While the 5-HTTLPR polymorphism and serotonergic responsivity in the fenfluramine challenge have been most robustly associated with poverty, other features of serotonergic neurotransmission appear to also be altered by poverty. For instance, Brummett et al. (2010) showed that the interaction between low socio-economic status and disease may be mediated by the serotonin metabolite 5-HIAA: these authors found that low-SES subjects have higher levels of the beta2-integrin CD11b , which is involved in the development of atherosclerosis; however, this was only true in subjects with low circulating levels of 5-HIAA. Thus lack of serotonin may facilitate the association between low SES and stress-related disease.

Jokela et al. (2007) examined the relationship between parental socioeconomic status, adult harm avoidance, and a particular polymorphism of the serotonin receptor 2A gene. Harm avoidance is one of four fundamental temperament traits in Cloninger's (1987) biosocial model of personality, and refers to behavioral inhibition, fearfulness, and cautiousness (Cloninger et al., 1987), and emotional and physiological stress reactivity (Puttonen et al., 2005). A number of studies have shown that harm avoidance is related to individual serotonin levels (Peirson et al. 1999, Hansenne and Ansseu 1999, Munafo et al. 2005), and in particular the binding potential of 5-HT2A receptors (van Heeringen et al. 2003). The binding potential of these receptors is in turn associated with the T102C polymorphism of the HTR2A gene (Turecki et al. 1999; see also Polesskaya and Sokolov 2002). Jokela found that indeed this polymorphism is related to harm avoidance , with the C/C genotype showing higher harm avoidance than the other genotypes. Interestingly, this relationship was mediated by parental SES: individuals with higher parental SES had lower adulthood harm avoidance, but this was only true for individuals carrying the T/T or T/C genotype and not those carry-

ing the C/C genotype. The authors speculate that the C/C genotype might make the 5-HT2A receptors less sensitive to environmental influences. This finding is in line with another study by the same authors, which showed that carriers of the C/C genotype showed no association between maternal nurturance in childhood and depressive symptoms in adulthood, while carriers of the other genotypes did show this association.

2.6 Serotonin and Economic Choice

In the previous section, I reviewed evidence showing that poverty is characterized by altered serotonergic neurotransmission. This section summarizes a growing body of literature suggesting that serotonin also plays a critical role in decision-making. In reviewing this literature, I focus on those studies that implicate serotonin in economic choice in particular; specifically, serotonin has been linked to impulsitivty and time- and risk preferences. Together with the link between serotonin and poverty, this body of evidence suggests that poverty may perpetuate itself by altering serotonergic neurotransmission and thereby inhibiting prudent economic choice.

I described above that poverty may reduce serotonergic responsivity as assessed by the fenfluramine challenge, and that this effect may be mediated by the 5-HTTLPR polymorphism: poor individuals show reduced prolactin levels in the fenfluramine challenge, and this effect is particularly strong in individuals carrying the S-allele of the transporter polymorphism. Do lower serotonin levels, in interaction with the S allele of the serotonin transporter polymorphism, also lead to changes in economic behavior? A growing number of studies indicate that this is indeed the case.

2.6.1 Evidence from Typtophan Depletion

The standard method to experimentally produce conditions of reduced serotonergic neurotransmission is dietary tryptophan depletion. Tryptophan is the precursor amino acid from which serotonin is synthesized; it is ingested with the diet, and therefore dietary interventions can be used to alter levels of tryptophan and thereby serotonin. Specifically, in tryptophan depletion, subjects are given an amino acid cocktail that either contains tryptophan or does not. In the latter case, plasma and brain levels of tryptophan, and thereby serotonergic function, are significantly reduced (Reilly, 1997). Thus, tryptophan depletion is a powerful experimental tool to induce reduced serotonergic function.

In an early study of the effect of tryptophan depletion on economic choice,

Rogers et al. (1999) tested subjects on the so-called Decision-Gamble task. In this task, subjects are presented with 10 red and blue boxes, and are asked behind a box of which color they think a yellow token is hidden. They are rewarded for predicting the location of the token correctly. This task has a normatively optimal choice of color, namely the color of the majority of the 10 boxes. The authors assessed the effect of acute TRP depletion on performance of this task. They found that tryptophan-depleted subjects showed worse performance, i.e. they chose the more likely outcome (the "correct" choice) on a smaller number of trials compared to controls.

In contrast, Talbot et al. (2006) found that TRP depletion actually improved decisions, i.e. TRP-depleted subjects chose the optimal action more frequently. It remains unclear what accounts for this difference. Talbot et al. (2006) also assessed risk-taking, which was quantified as the proportion of points bet in the gambling task, and impulsivity, quantified as the difference in the decisions in the ascending vs. descending sequences of displays of possible bets (this can be understood as an aversion to waiting for the desired bet to be presented). They found no effect on these tasks. Note, however, that these tasks are not what economists usually understand as risk-taking tasks; the proportion of points bet is only a measure of risk-taking if one takes into account the probabilities involved, and the difference between choices on the ascending and descending bet presentations might also be noise, failure to maximize reward, or disinterest in the task.

A partial resolution to this contradiction was obtained by Rogers and colleagues (2003). These authors presented subjects with a gambling task in which they chose between a "control gamble", with a 50-50 chance of winning 10 vs. losing 10 points, and an "experimental gamble" which varied in terms of probabilities and stakes, with high or low probabilities (25% vs 75%) and high or low stakes (80 vs. 20 points). In addition, a standard risk-aversion trial type presented subjects with a choice between a certain 40 point gain vs. a 50-50 chance of winning 80 points or 0 points, and a loss-aversion trial type presented subjects with a choice between a certain 40 point loss vs. a 50-50 chance of losing 80 points or 0 points. Rogers and colleagues found that participants chose the experimental gamble more frequently when its expected gains were large compared to when they were small; however, this effect was less pronounced under TRP depletion. Given that both the control and the experimental gambles involved an element of risk, it is difficult to interpret this pattern as a change in risk aversion; in addition, the standard risk-aversion trials described above showed no effect of TRP depletion: subjects chose the safe gambles more often than the risky gambles, and this effect did not differ across TRP+ and TRP- groups. The authors conclude that TRP depletion attenuates subjects' ability to discriminate between gains of

different expected magnitudes. An alternative explanation is that Rogers and colleagues found no effects of TRP depletion on risk preferences because the task was not incentivized.

Murphy et al. (2003) used the same task as Rogers et al. (2003), combined with dietary tryptophan supplements (rather than depletion, as in the previous study). The authors found that tryptophan supplements exacerbated the reflection effect in risky choice. This effect consists in risk aversion in the gain domain, and risk-seeking in the loss domain. After receiving tryptophan supplements, this effect was attenuated: TRP-treated subjects chose the risky option more often in the gain domain, and less often in the loss domain, compared to controls, indicating that increases in serotonin availability led to less risk aversion in the gain domain and less risk seeking in the loss domain. By implication, the absence of serotonin would predict increases in the reflection effect, i.e. more risk-aversion in the gain domain and more risk-seeking in the loss domain. However, tryptophan depletion in the previous study by the same authors did not produce these results.

Finally, converging evidence on the role of serotonin in risky choice comes from primates: Long et al., (2009) presented macaque monkeys with a risk preference task, where they could choose between a safe and a risky option. When the two options had expected values, any significant preference for the safe option can be interpreted as risk aversion. The authors found that tryptophan depletion decreased monkeys' preference for the safe option, i.e. made the monkeys more risk-seeking. When the expected values differed, the relative preference for the safe vs. risky option can be interpreted as the risk premium, i.e. the amount of extra juice required by the monkeys to choose the risky over the safe option. In line with the results obtained for equal expected values, under tryptophan depletion monkeys had lower risk premia than otherwise, suggesting again that lower serotonin decreased risk aversion. This is in contrast to the human studies discussed above, in which decreased risk aversion (at least in the gain domain) was seen for increased levels of tryptophan (Murphy et al., 2003) were generally associated with more risk-averse choice.

2.6.2 Evidence from Genetics

As discussed in the previous section, poverty may interact with the serotonin transporter gene 5-HTT to affect serotonergic responsivity; carriers of the short allele of the 5-HTTLPR polymorphism have a blunted response in the fenfluramine challenge, but this effect is stronger in poor compared to rich people. In other words, poverty exacerbates the effect of this polymorphism on serotonergic neurotransmission. This raises the question whether the short

allele of this polymorphism may have behavioral consequences which might in turn contribute to exacerbating poverty. Indeed, it will emerge in the following discussion that the short allele has indeed been shown to affect economic choice, in potentially adverse ways.

On a general level, the 5-HTTLPR polymorphism appears to be associated with impulsivity; for instance, Sadeh and colleagues (2010) observed a main effect of 5-HTTLPR genotype on the impulsivity dimension of psychopathology, with SS individuals exhibiting greater impulsivity than LL carriers. In addition, however, this effect appears to be exacerbated by low SES: In a sample of Italian adolescents, Nobile et al. (2007) showed that low SES interacted with DRD4 long and 5-HTTLPR long alleles to produce higher aggressive behavior scores on the Child Behavior Checklist/6-18. In other words, low-SES individuals who carried both long DRD4 and long 5-HTTLPR alleles scored higher on this measure of aggression.

In the Iowa Gambling Task described above, Must et al. (2007) found that subjects chose the disadvantageous decks more frequently if they were SS compared to LL carriers. The subjects in this study all suffered from major depressive disorder (MDD), thus it is not clear to what extent their finding generalizes to normal volunteers. However, a number of studies have since refined and extended this finding: in particular, evidence is emerging that subjects carrying the SS allele may be slower at learning to choose from the optimal decks in the IGT than LL carriers. For instance, Jollant and colleagues found that subjects carrying two long or one long and one short allele of the 5-HTTLPR gene improved their performance over time while performing the IGT, whereas subjects with two short alleles showed no such improvement. Unfortunately the authors do not report whether the interaction effect of time x genotype on performance is significant; in addition, their subjects all had a history of suicide attempts and are therefore not entirely representative. However, in a similar study in obsessive-compulsive patients, da Rocha et al. (2008) found that LL-carriers showed better learning as the IGT progressed compared to SS and SL carriers. Moreover, Homberg and colleagues (2008) obtained similar results in normal subjects, showing that women who carried two S alleles were worse at learning to respond optimally in the IGT that those who carried at least one L allele. The authors interpreted this finding as an indication that lack of serotonin causes subjects to maintain the established choice option; however, the possibility that they had a general learning impairment remains open. Regardless, the S allele appears to impair optimal actions in a simple economic maximization task. A study by Ha et al. (2009) qualifies this conclusion somewhat, as these authors failed to find a main effect of 5-HTTLPR genotype on performance; instead, they found that the 5-HTTLPR polymorphism interacted with a dopaminergic

polymorphism, namely widely studied dopamine receptor D4 polymorphism (DRD4): among subjects who carried the short allele of 5-HTTLPR, those who also carried the 2R- allele of the DRD4 polymorphism performed worse at the IGT than those who carried the 2R+ allele; in contrast, among those who carried the long 5-HTTLPR allele, 2R- carriers performed better than 2R+ carriers. These findings confirm the involvement of the 5-HTTLPR polymorphism in economic choice, whnlie in addition suggesting that the details of how this influence interacts with other neurotransmitter systems remain to be clarified.

While the Iowa Gambling Task measures to what extent subjects are capable to optimizing their behaviour to maximize reward, it does not reveal the effect of genetic variation on economic preferences (arguably the IGT contains an element of time preference; however, since the timing of payment and consumption do not differ for the early vs. late trials in an experiment, this claim is difficult to support.) Could genetically modulated serotonergic neurotransmission also affect economic preferences? To answer this question, Kuhnen & Chiao (2009) presented subjects with a financial risk-taking task, in which they could allocate investments between a riskless and a risky asset. The subjects could allocate $23 or $28 between these two assets. The riskless asset paid a known rate of return with certainty, while the riskless asset paid either of two rates of return with equal probability. One trial was chosen randomly for payment. Risky investment was measured by the difference between the amount each individual participant invested in the risky asset, and the amount invested in the risky asset by the average participant.

The authors found that individuals carrying two S alleles of the 5-HT transporter gene were significantly more risk-averse than those with S/L or L/L genotypes, in that they invested 28% less in the risky asset than the other participants. The authors speculate that the long allele may confer novelty-seeking behavior that is evolutionarily adaptive.

In a study notable for its completeness and interesting results, Crisan et al. (2009) extended the role of 5-HTTLPR genotype to a number of other economically relevant behaviors. First, the authors found that S-allele carriers showed increased fear conditioning compared to LL individuals: after watching a movie in which another person was conditioned to associate the presentation of squares of a certain color with electric shocks, subjects showed an increased skin conductance response to those colors that had been paired with shock in the observed participant. (Note that participants never actually experienced shocks themselves, making it slightly ambiguous through what channels this learning effect operated.) Notably, this increase in SCR to the CS+ compared to CS- was larger in subjects carrying at least one S-allele.

Second, the authors confirmed the involvement of the 5-HTTLPR poly-

morphism in risk preferences by tested risk taking in the Balloon Analogue Risk Task (BART). In this task, subjects are asked to pump up a balloon on a computer screen to accumulate rewards. However, if the balloon is pumped up too much, it pops, and the accumulated points are lost. The authors found that S allele carriers were significantly more risk-averse, i.e. stopped pumping at smaller balloon sizes, than LL individuals.

Similarly, S-allele carriers showed a larger reflection effect in risk-taking. Using the framing task proposed by De Martino et al. (2006), the authors found that the difference between risk-aversion in a gain frame and risk-seeking in a loss frame was greater in these subjects than in LL carriers. (The authors do not report the interaction term, but the results appear solid.) Interestingly, this effect was entirely driven by increased risk-seeking in the loss frame in S-allele carriers, while risk aversion in the gain frame showed no effect of genotype. This contrasts with the BART findings above, potentially because the BART task is more experiential than the somewhat abstract financial risk task.

The findings of Crisan et al. (2009) were confirmed by a similarly elegant study by Roiser et al. (2009), which combined genetic information with fMRI data. Presenting subjects with the same framing task as described above, these authors confirmed the larger behavioral framing effect in SS compared to LL subjects. In addition, they found that amygdala activation during task performance differed between SS and LL individuals: in particular, the authors observed a genotype x frame x decision interaction on amygdala activation: in SS participants, amygdala activation was higher when subjects chose according to frame than when they chose counter to frame, i.e. when they chose the gamble option in the loss frame or the safe option in the gain frame. This interaction was not seen in LL subjects.

The authors then performed a psychophysiological interaction analysis (PPI, Friston et al., 1997). This analysis allows to assess coupling between a brain region of interest and other regions depending on experimental context. In this case, the authors were interested in the coupling between the amygdala and other brain regions when subjects chose according to frame vs. counter to frame, and in whether this coupling varied as a function of genotype. Indeed, they observed that the coupling between anterior cingulated cortex (ACC) and amygdala was modulated by subjects' decisions: connectivity was high when decisions were counter to frame, but low when decisions were in line with frame. However, crucially this effect was only observed in LL individuals; SS individuals showed low connectivity between these regions regardless of whether their decisions were made in accordance with or counter to frame. The authors speculate that the ACC may have a role in overriding emotional responses represented in the amygdala, and that this inhibition

might be attenuated in SS individuals. These findings are in line with the involvement of amydala and ACC in serotonin-modulated behaviors outlined above.

In sum, it appears that the short allele of the 5-HTTLPR polymorphism indeed affects economic choice: it impairs optimal decision-making in the Iowa Gambling Task, and leads to risk aversion and increased reflection effects in financial decision-making. The fact that the effects of this allele on serotonergic neurotransmission are exacerbated in poor individuals suggests that poverty may perpetuate itself by affecting serotonergic neurotransmission through the 5-HTTLRP polymorphism, which in turn affects economic choice behaviour and thereby potentially reinforces poverty.

2.7 Testosterone and Poverty

2.7.1 Testosterone Basics

Testosterone is one of the body's major sex hormones. A steroid hormone like cortisol, it occurs in both men and women; in men, it is mainly produced by the Leydig cells of the testes, while in women it is produced by the ovaries and placenta. In both sexes, it is also produced by the adrenal cortex. Secretion is regulated by the hypothalamic-pituitary-testicular (HPT) axis: the hypothalamus releases gonadotrophin-releasing hormone (GnRH), which in turn causes the release of lutenizing hormone (LH) and follicle-stimulating hormone (FSH) by the pituitary gland, which then leads to testosterone production in the testes (men), and estrogen production in the ovaries (women). In the brain, it acts on structures that are crucial for emotion regulation, memory, and decision-making, such as amygdala, hippocampus, and prefrontal cortex (McEwen & Milner, 2007; de Kloet et al., 1998).

2.7.2 Testosterone and Poverty

The evidence on the linke between poverty and testosterone levels is weaker than in the case of cortisol and serotonin. Nevertheless, a number of papers have reported associations between baseline testosterone levels and SES.

In the largest and most persuasive study to date, Dabbs & Morris (1990) studied a sample of 4,462 male U.S. military veterans. They classified those whose testosterone levels were in the highest 10% of the distribution as being high in testosterone. In self-report measures, these individuals reported more excessive and antisocial behaviors: the were more likely to be delinquent, to abuse drugs and alcohol, to have many sex partners, and to have gone

"AWOL" (absent without leave) in the military. Dabbs & Morris then divided the sample into high- and low-SES groups (defined as being above vs. below the US median in both income and education). Two findings emerged. First, the low-SES group had higher levels of testosterone: 14% of the low-SES group were in the top 10% in terms of testosterone levels, while only 6% of the high-SES group were in this group. In addition, the relationship between testosterone and antisocial behaviors was mediated by SES: the low-SES group showed significant relationships between testosterone levels and the antisocial behaviors mentioned above, while no such relationship was evident in the high-SES group. A potential confound is sample size, in that the high-SES group had fewer individuals in the upper 10% of the testosterone distribution than the low-SES group, namely only 73. However, this finding nevertheless suggests a link between low SES and high testosterone levels.

In a later paper, Dabbs (1992) related the serum testosterone levels of the same group to their occupational status, and found that blue-collar workers and the unemployed had higher testosterone levels than white-collar workers. Controlling for age and race did not alter these results. In addition, among employed participants, testosterone levels correlated negatively with Sevens & Cho's (1985) occupational status score. (Intriguingly, farmers had some of the lowest testosterone levels, even though their occupational standing and income was likely more comparable to that of blue-collar than white-collar workers.) Dabbs shows that the correlation between testosterone and occupational status may be mediated by intelligence, antisocial behavior, or education. More generally, the direction of causality remains unclear; Dabbs argues that testosterone may be high in childhood due to its high heritability (Meikle et al., 1987, 1988) and subsequently affect intelligence, education, and antisocial behavior. However, environmental conditions associated with low SES may have direct effects on testosterone levels as well, as outlined below.

Gray et al. (2006) compare baseline testosterone levels among five different population strata studied in the THUSA (Transition and Health During Urbanization of South Africans) study (Vorster et al., 2000). In this study, a sample of 1854 respondents from the North West Province of South Africa was classified according to urbanization: rural groups were those living in tribal areas and people living on commercial farms, while urban groups were those living in informal settlements ("squatter camps"), those living in established townships with access to water and electricity, and finally those living in western-style houses in upper-class suburbs. Gray et al. found that the last of these groups, i.e. fully Westernized participants from affluent suburbs, had significantly higher testosterone levels than the other groups. However, this difference was no longer significant after potential confounds

were controlled for, namely physical activity levels, depression, affect, and hostility.

Two further studies from the same author provide related anecdotal evidence. First, Dabbs et al. (1990) studied testosterone levels in seven occupational groups; these authors found that ministers had lower testosterone levels than actors or football players, but it remains unclear to what extent this difference is related to occupational status, income, or other variables, and moreover whether it is a result or a consequence of any of these variables.

Second, Dabbs et al (1998) suggest that trial lawyers are the "blue-collar workers" of the legal profession, in that they engage directly with defendants and compete forcefully with one another. They find that, indeed, trial lawyers have higher levels of testosterone than other types of lawyers such as patent lawyers.

Together, these results suggest that poverty and low socioeconomic status may be associated with higher levels of testosterone. As in the previous sections, concerns remain about the direction of causality of this relationship. Nevertheless, at least the correlation appears to exist. Equipped with this finding, we can now ask to what extent these raised testosterone levels in poverty have behavioral consequences.

2.8 Testosterone and Behavior

Testosterone has a prominent role in controlling behavior. It is involved in regulating sex drive, muscle development, and the behavioral fight-or-flight response; in addition, it has been closely linked to dominance- and status-related behaviors (Mazur & Booth, 1998) and aggression (Book et al. 2001). In particular, it appears to increase competition (Silverin, 1980; Hegner & Wingfield, 1987), lead to confrontational responding in status and dominance challenges (Muller & Wrangham, 2004; Dabbs, 1997), and contribute to establishing and maintaining social hierarchies (Dugatkin & Druen, 2004). Thus, testosterone's role in behavior appears to be one of signaling and regulating social status and the response to status challenges (Mazur & Booth, 1998). In the following, I disucss three mechanisms by which testosterone interacts with environmental factors to produce such behaviors in btoh adaptive and maladaptive fashions.

2.8.1 The Winner Effect

One of the most established findings regarding the behavioral effects of testosterone is the so-called "winner effect". The winner effect refers to two fre-

quently replicated findings. First, testosterone levels rise in anticipation of an antagonistic or dominance encounter or a status challenge; second, after the challenge, levels rise further in winners, but decline in losers. These differences in post-encounter testosterone levels then contribute to further success in winners, and further losses in losers. Thus, the winner effect describes a self-reinforcing cycle in which positive testosterone feedback leads winners to keep winning and losers to keep losing.

For instance, Bernstein et al. (1974) introduced 5 male macaques to a group of 34 other macaques. The newcomers were quickly attacked and defeated by the resident group, and this event was accompanied by an 80% drop in their circulating testosterone levels. A different setting that allowed the intruder to achieve victory over the resident group led to strongly increased testosterone levels in this animal.

A similar effect was shown in humans by Booth et al. (1989), who found that tennis players who won a match had rising testosterone levels throughout the match, while those who lost showed declining levels. Similarly, Mazur & Lamb (1980) found that winning players had higher levels after the match than losers.

A potential concerns with these findings is endogeneity, i.e. players might have won *because* of their high testosterone levels, rather having had high testosterone as a consequence of their wins. To alleviate this concern, Gladue et al. (1989) experimentally manipulated winning vs. losing by randomly assigning participants to one or the other outcome in a laboratory game. As in Booth's study, winners had higher testosterone (but not cortisol) levels compared to losers. In line with these results, recently released hostages from Iraq showed strongly elevated levels of testosterone, possibly as a result of elation over their release (Rahe et al., 1990). In contrast, Mazur & Lamb (1980) did not observe such a difference when winning vs. losing was determined by a lottery draw; thus, testosterone surges after winning experiences appear to be restricted to situations where winners attribute the win to their own actions.

In a similar experiment with non-endogenous winning vs. losing outcomes, McCaul et al. (1992) placed subjects in a situation in which they could win or lose $5, the outcome being governed entirely by luck. Intriguingly, the authors again found that testosterone levels were higher in winners than in losers, suggesting that testosterone may respond to status-altering outcomes. McCaul and colleagues also measured cortisol, but found no effect of winning vs. losing on this measure.

Finally, Bernhardt et al. (1998) used a natural setting which arguably controlled for the endogeneity of winning vs. losing outcomes to a similar extent. These authors studied the testosterone levels of football fans before

a World Cup match between Brazil and Italy. These fans did not expect to actively participate in the contest themselves, and thus changes in their testosterone levels must reflect the anticipation of a status encounter and the experiences of winning and losing. Indeed, Bernhardt et al. found that testosterone levels were elevated in anticipation of the match in both groups of fans. In addition, fans of the winning team, Brazil, showed increased testosterone levels after the match, while Italy fans showed decreased levels.

A potential concern with all of these findings is that even in those studies in which winning vs. losing was manipulated by the experimenters (or, in the acse of the World Cup study, by the relative performance of two teams which were closely matched in terms of ability), participants might have come to the experiment with home-grown beliefs about their own likelihood of winning; in this way, changed testosterone levels after wins vs. losses might relfect the degree to which subjects' expectations regarding their own performance were fulfilled, as opposed to winning vs. losing experiences per se.

Importantly, the changes in testosterone levels as a result of winning vs. losing also have further behavioral consquences, namely in that winning individuals with higher testosterone levels are more likely to win subsequent status challenges than losers with lower levels. For instance, Oyegbile & Marler (2005) staged fights between male mice; in an initial phase, "winners" were created among the test animals by pairing them with weaker and mildly sedated opponents. The animals won most of these fights; based on the above findings, one would therefore expect a testosterone surge after such wins. Indeed, after two wins, the winners showed increased testosterone levels compared to control, and after three wins, they were more likely to win subsequent fighting encounters than under control conditions. Similar findings have been produced in a number of other species: insects (Otronen, 1990; Whitehouse, 1997), fish (Hsu & Wolf, 1999), and reptiles (Schuett, 1997). While no demonstration of the effect appears to exist in humans, the animal literature strongly suggests that prior wins indeed contribute to subsequent wins, and that this effect is mediated by the testosterone surge that follows winning experiences.

Together, these studies suggest an important potential channel through which the effects of testosterone on behavior may amplify themselves: individuals with high testosterone are likely to win dominance encounters; this further raises testosterone levels, and these raised levels in turn contribute to subsequent wins. Conversely, losers will show a "loser effect", in which losing a dominance challenge leads to decreased testosterone levels and subsequently a higher probability of losing dominance encounters.

2.8.2 The Mismatch Effect

A second strand of literature modulates these winner effect findings in an intriguing way. The winner effect refers to rises in testosterone levels in anticipation of status or dominance challenges, and further rises after a win, thus leading to a cycle in which one win leads to subsequent ones, and one loss leads to further losses. However, a second group of studies describes a parallel finding known as the Mismatch Effect. The basic claim of this literature is that the same testosterone level may operate in different ways in different situational contexts. Specifically, high-testosterone individuals may improve in performance when they find themselves in dominant positions; in contrast, when they find themselves in submissive positions, their performance may deteriorate.

Josephs et al. (2006) placed male participants with either high or low baseline testosterone levels in a rigged competition, in which they completed cognitive tasks such as spatial processing. By randomly letting one participant win, Josephs et al. manipulated relative status. They then assessed cognitive functioning with questions from the analytic section of the Graduate Record Examination (GRE). It turned out that participants with high baseline testosterone performed best when they were in the "dominant" position after the rigged competition, while low-testosterone individuals performed best when they were in the "submissive" position. The authors suggest that when there is a mismatch between one's desired status, as proxied by baseline testosterone levels, and actual status, as assigned in the rigged competition, participants are distracted by this mismatch and therefore perform worse on the cognitive task. This finding would imply that low-status individuals with high testosterone will perform poorly. If, in addition, low status has environmental consequences which increase testosterone, this could lead to a vicious circle by which an environment of poverty leads to high testosterone, and in combination with awareness of one's low status, this leads to impaired performance and antisocial behavior.

These findings suggest an additional channel to the winner effect by which poverty may affect testosterone levels and behavior: in the winner effect model, a fall into poverty would decrease testosterone levels, lead to subsequent losses, thereby further decreasing testosterone, and so on. In the mismatch effect model, a fall into poverty would interact with previous testosterone levels to produce adaptive behavior in individuals with previously low testosterone, and maladaptive behavior in individuals with previously high testosterone. Thus, if poor people have initially high testosterone, an increase in poverty would lead to a "mismatch" between with their low social status and their high testosterone levels, and thus lead to bad performance, which

would further exacerbate poverty. Thus, the mismatch effect can produce a self-perpetuating cycle in which poverty reinforces itself.

Note, however, that the winner and mismatch effects operate in opposite directions: according to the mismatch effect, individuals with high testosterone who find themselves in a low-status position should perform badly, and therefore descend in the hierarchy. This is consistent with the correlational findings mentioned above. However, the winner effect would suggset that over time poor individuals should lose more dominance challenges than richer people and therefore show *lower* testosterone levels, while rich people would be more likely to win status challenges and therefore should show higher levels.

How can these contradictory findings be reconciled? First, note that the winner and mismatch effects are not mutually exclusive: on the one hand, high-testosterone individuals may perform poorly when placed in situations in which they are the "underdog" (mismatch effect); on the other hand, according to the loser effect, such poor performances would lead to lower testosterone levels, which in these situational contexts would produce adaptive behavior, etc. Thus, what is necessary to create a vicious cycle in which high testosterone in poor people leads to maladaptive behavior and thus further poverty, is that the mismatch effect is stronger than the winner effect. I am unaware whether this proposition has been tested; it is interesting material for future studies.

A further problem is that according to the above argument, rich people with low testosterone levels would find themselves in a mismatch between their high social status and their low testosterone levels, which, according to Josephs' findings, should also lead to maladaptive behavior, and thus a descent in the hierarchy. However, an additional line of evidence argues against this relationship.

A modified version of the mismatch argument is that the behavioral effects of testosterone may only come to bear in situational contexts in which one's status is threatened. According to this line of reasoning, rich people may find themselves in environments where they do not encounter frequent status threats, and for this reason the mismatch between their low testosterone levels and high status does not lead to maladaptive behavior.

In support of this argument, such a context-related effect of testosterone on performance was found by Josephs et al. (2003). These authors primed gender stereotypes for performance on math questions, by presenting participants with a questionnaire asking questions such as "I think that some people feel I have less math ability because of my gender". The participants then responded to items from the quantitative section of the GRE (GRE-Q) for 20 minutes. The authors found that men with high testosterone, for whom

the math test offered the possibility to confirm a positive stereotype, performed well on the GRE-Q, while women with high testosterone, for whom the math test would be more likely to be seen as a status threat due to negative stereotypes about the mathematical ability of women, performed poorly. Thus, high testosterone appears to impair performance in contexts where one is faced with status threats.

Further evidence for this view comes from a study by Millet & Dewitte (2007). These authors found that the effect of testosterone on aggression is mediated by situational context. Rather than measuring testosterone levels directly, these authors used a simple indicator of prenatal testosterone exposure: the 2D:4D ratio, i.e. the ratio of the length of the index compared to the ring finger. A low 2D:4D ratio has been shown to be associated with high prenatal levels of testosterone, while a high ratio is thought to reflect low levels (Csatho et al., 2003; Manning, 2002; Manning et al., 1998; Williams et al., 2003).

Millet & Dewitte found that subjects with high testosterone levels (lower 2D:4D ratio) scored higher on questionnaire measures of aggression (Buss & Perry, 1992; O'Connor et al., 2001). However, this relationship obtained only after participants had viewed a violent video, not after viewing neutral videos. Thus, situational context appears to uncover the effect of testosterone on aggressive behavior.

Together, these findings suggest that the mismatch effect may not necessarily make rich people poor over time due to the contrast between their high social status and low testosterone levels: it may be that the relationship between testosterone and behavior only becomes apparent when status is threatened, and rich people may only rarely find themselves in such situations.

2.8.3 Testosterone and Economic Choice

I have argued above that poverty may lead to increased testosterone levels, which in turn may interact with social status to produce suboptimal behavior. However, the behaviors in question were cognitive performance; I have ignored evidence on the effect of testosterone on economic choice so far. In fact, the evidence is mounting that testosterone has far-reaching consequences for economic behavior.

2.8.4 Testosterone and Time Preference

A small group of studies suggests that high testosterone may be associated with present-biased economic decisions. Wilson & Daly (2004) provide early

anecdotal evidence for this view: these authors presented men with pictures of attractive women; it was thought that viewing of attractive female pictures would increasetestosterone levels, and so this paradigm arguably offers a non-invasive testosterone manipulation. Wilson & Daly observed that this manipulation increased temporal discounting, i.e., made men more impatient, than viewing control pictures.

In a purely correlational study, Takahashi et al. (2006) measured the association between baseline testosterone and the discount parameter k (Kirby et al., 1999). No linear relationship was found, but a regression that included a quadratic regressor for log(k) resulted in significance for all coefficients. Specifically, testosterone increased as discounting increased for subjects with low k values (i.e. patient subjects), while testosterone decreased as discounting increased for subjects with high k values. It is somewhat unclear as to how this finding is to be interpreted, mostly because it is more intuitive to think of discounting as a consequence of testosterone levels, but if one views it as such no clear relationship is apparent. In a partial answer to this puzzle, other studies have found that the relationship between testosterone and discounting may be mediated by social status.

Specifically, Kobe & Millet (2004) capitalized on this fact to study the relationship between baseline testosterone and economic choice under difference social status conditions. They manipulated social status in the lab by asking participants to work on a Sudoku puzzle, and then providing bogus feedback on their performance. In the high-status condition, participants were told that they had performed the task better than 90.7% of the participants in a previous study, while in the low-status condition, they were informed that their performance only put them at the 11.4 percentile. Participants then completed a time discounting task, in which they indicated which amount of money they would require after a certain delay to make them indifferent between that amount and 15 Euros on the day of the experiment. Kobe & Millet found that testosterone was related to discounting, but this relationship was mediated by status assignment: participants in the low-status condition showed a relationship between 2D:4D ratio and discounting, in that a smaller 2D:4D ratio (and thus, higher testosterone) was associated with higher discounting; in contrast, in the high-status condition, no such relationship existed. Thus, higher testosterone levels may lead to impatience in low-status, but not in high-status conditions; put differently, a low-status environment may allow the effects of testosterone on impatience to come to the fore, while these effects are suppressed in a high-status environment. A potential problem with the 2D:4D metric is that it is somewhat unclear how high vs. low prenatal testosterone levels play out later in life. On the one hand, the 2D:4D ratio correlates with circulating hormone levels (Falter

et al., 2006; Manning et al., 1998); on the other, it is also associated with testosterone responsivity (Manning et al., 2003). Thus, direct measures of circulating levels will be required to rigorously establish the link suggested by this intriguing study.

2.8.5 Testosterone and Risk Preference

In addition, a growing number of studies implicate testosterone in risk-taking behavior. For instance, Apicella et al. (2008) had subjects play a simple investment game to measure their risk preferences: subjects were asked to decide what fraction of $250 they wanted to invest in a risky asset. The payoff from the risky asset was determined by a coin flip; it either paid 0, or a return of 250%, with equal probability for both outcomes. The expected value of investing the entire $250 is $325.50; thus, expected-value maximizing subjects should invest the entire amount. The fraction of the initial endowment that is not invested is therefore a measure of subjects risk aversion. Apicella and colleagues found that subjects with higher baseline testosterone levels showed less risk aversion, i.e. invested a higher fraction of their endowment in the risky asset. The same was true for subjecst with higher facial masculinity scores. Facial masculinity is a measure of testosterone exposure during puberty, and reflects the fact that high testosterone exposure during this period leads to particular facial features such as high jawbones and low cheekbones (Johnston et al., 2001).

Coates & Herbert (2008) tested whether testosterone correlated with economic performance of traders on a London trading floor. As described above for cortisol, these authors measured salivary cortisol and testosterone levels in a sample of 17 traders from a London trading floor twice daily, at 11am and 4pm. The cortisol results have been described above. The testosterone results revealed a correlation between testosterone levels and Profits and Losses (P&L) at the end of the day, i.e. 4pm. Crucially, both the 11am and the 4pm testosterone levels correlated with P&L independently; this suggests, on the one hand, that higher morning testosterone levels predict higher profits during the remainder of the day; and, on the other hand, that higher profits during the day raise testosterone levels in their turn. Thus, these traders show something like a winner effect in the relationship between their testosterone levels and their profits: higher testosterone leads to higher profits, which in turn raises testosterone levels further. It is likely, however, that further rises in testosterone levels may lead to adverse outcomes; for instance, Booth et al. (1999) found that high-testosterone men showed a higher incidence of behaviors that are harmful health-related behaviors, Daitzman & Zuckerman (1980) found a correlation between high testosterone and impulsive behavior.

Reavis & Overman found a correlation between baseline testosterone levels and performance on the Iowa Gambling task: college-age men with higher baseline testosterone levels performed worse than those with lower levels. However, this finding was only significant in college-age men, not in older men or women.

To avoid the correlational nature of such findings, a number of studies have manipulated testosterone pharmacologically in health participants to study its effecst on economic choice. For instance, using chronic testosterone administration over a period of 4 weeks, Zethraeus et al. studied the effect of an exogenous testosterone increase on economic behaviors in a sample of 200 postmenopausal women. No effects of testosterone were found on a host of studied behaviors, including risk attitudes, altruism, and trust and trustworthiness; however, it is possible that this was due to the fact that acute administration has been shown to have stronger effects on behavior.

Van Honk et al. (2004) made this correction: they administered a single dose of testosterone to participants who subsequently played the Iowa Gambling Task. Performance on the task was impaired under testosterone compared to placebo, with participants on testosterone choosing cards from the disadvantageous deck more frequently than under placebo conditions.

Thus, it appears that high testosterone leads to present-biased time preferences and risk-seeking risk preferences. To what degree this finding can be reconconciled with the literature cited for cortisol above remains to be seen; in particular, on the one hand, poverty is associated with higher cortisol and more risk aversion, while on the other hand povery is also associated with higher testosertone and *less* risk aversion. Future studies will have to elucidate which of these effects dominates, and under which conditions.

2.9 Emerging questions

2.9.1 What about causality?

While laboratory experiments such as that of Porcelli & Delgado (2009) have already begun to identify casual effects of neurobiological variables on economic choice, it remains unclear whether and to what extent the link between poverty and neurobiological outcomes is causal: to date, all of these relationships are purely correlational, and reverse or simultaneous causation are therefore serious concerns. Future RCTs should make it a priority to measure the effect of poverty alleviation, or exogenous increases in poverty, on levels of cortisol, serotonergic responsivity, and serotonergic gene-environment interactions. In the following, I outline three potential approaches.

A laboratory approach

A first approach to asking whether neurobiological variables are affected by poverty is to induce a version of poverty in a laboraty setting, and then measure its impact on neurobiological variables and economic choice. This may initially appear to be a hopeless proposition; poverty is an extremely multi-faceted phenomenon, which, apart from insufficient income, encompasses poor access to health facilities, low levels of education, poorly developed institutional environments, etc. Thus one naturally cannot hope to capture the full range of what it means to be poor through a simple laboratory manipulation. At the same time, however, experimental economics has a history of successfully operationalizing complex real-life behaviors in the laboratory; consider for instance the trust game, which is an established laboratory measure of trust, and the dictator game, a widely-used measure of altruism (Bolton et al., 1998; see Camerer, 2003, for an overview). Thus, it is likely that at least some aspects of poverty can be captured in a laboratory situation. Indeed, the central defining element of poverty, namely the absence of money, should in principle be quite amenable to study in the laboratory.

In practice, one could experimentally manipulate both poverty relative to other individuals' income levels, and poverty relative to a subject's individual income history. The former could be achieved by giving subjects different initial endowments at the beginning of the experiment, and informing them about the endowments of other participants; thus, each participant knows how "poor" she is relative to others. The latter could be achieved by letting subjects earn income in the experiment, e.g. in a real effort task such as that used by Abeler et al. (2009), and then exogenously imposing income shocks on participants. For instance, a subset of participants might randomly lose a large proportion of their earned income, while another subset gains a comparable proportion, and yet other participants remain at the same level. One could then measure how these manipulations affect economic, psychological, and neurobiological outcomes; i.e., does being poor relative to one's own income history affect economic choice (e.g. time and risk preference, prosocial behavior, and effort levels), psychological variables (e.g. stress, locus of control, optimism, and self-esteem), and neurobiological outcomes (e.g. cortisol and serotonin levels, potentially in interaction with the polymorphisms mentioned above)? Despite the obvious limitations of a laboratory study such as this one, discussed above, the answers to these questions might at least provide suggestive evidence that could be compared to field data.

A poverty increase in the field

Due to the limitations of the laboratory approach, field evidence could potentially provide a much more satisfying answer to the causality question. Does an increase in real-world poverty lead to changes in psychological, economic, and neurobiological outcomes? Of course it is not possible to experimentally induce a downward income shock; however, it may be possible to identify natural experiments which have done just this. As an example, the Maasai in Kajiado district of Southern Kenya have experienced the worst drought in over a century in 2008/2009. This meant that almost all households in the region lost between 50-100% of their livestock. This was a terrible tragedy for the people in the region, but at the same time it affords an opportunity to study the effect of this negative income shock on welfare. How do we identify the causal effect of the drought on welfare? One possibility is to use an instrumental variables approach: in cases where detailed rainfall data are available, as is the case in this setting, this data can be used as an instrument for the number of cows owned by each household that died during the drought. One can then estimate the effect of this negative income shock on the psychological, economic, and neurobiological outcome variables of interest.

A poverty decrease in the field

Another possible approach to establish causality in the link between poverty and neurobiological outcomes, and one that is amenable to RCTs, is to test the psychological, economic, and neurobiological consequences of a *reduction* of poverty. The answers to this question could conceivably be quite different to those obtained for an increase in poverty; for instance, one could easily imagine that while an increase in poverty might lead to depression, poverty alleviation might not necessarily reduce depression that has already developed. As an example, imagine an RCT which distributes Unconditional Cash Transfers (UCTs) among poor residents in rural Kenya. An UCT is, in essence, "free money": a one-time, no-strings-attached payment to a particular household, based on basic criteria of need. From the point of view of our research question, namely, what is the effect of poverty alleviation on household welfare in general and neurobiological outcomes specifically, UCTs are an obvious vehicle of choice, since they represent the purest form of poverty alleviation: the central feature of poverty, as mentioned above, is lack of funds; UCTs address this characteristic head-on and thus offer the cleanest poverty manipulation for our purposes. In addition, UCTs are a promising new approach from a policy perspective, and initial natural experiments

(Duflo, 2000) and RCTs (Paxson & Schady, 2007) showed positive results. A concern in this literature is that UCTs are bound to have heterogeneous treatment effects; neurobiological measures of cortisol and serotonin could potentially detect aggregate effects of these effects, and thus offer a useful tool to assess the welfare consequences of UCTs. Conversely, UCTs offer an opportunity to cleanly test the effect of a poverty reduction on neurobiological outcomes.

2.9.2 How big are the effects?

Poverty traps require strong feedback loops to have explanatory power. Thus the question arises whether the neurobiological relationships described above are strong enough to account for the differences in economic choice observed between rich and poor people. For instance, how large are the poverty-related differences in cortisol levels that are typically observed between rich and poor individuals compared to stress-induced cortisol increases in the laboratory, and what proportion of the observed differences in economic choice between rich and poor could potentially be explained by this stress channel?

The problems described above make it difficult to answer this question. First, it is not clear to what extent laboratory-induced stress effects are similar in their behavioral effect to chronic cortisol differences; second, the question whether the link from poverty to increased cortisol levels is causal has yet to be answered conclusively. However, with these caveats in mind, we can at least describe an illustrative example to assess whether the relationships are of an order of magnitude that could potentially account for a satisfactory amount of the variance in economic choice between rich and poor.

Cohen et al. (2006a) contains the most detailed information on cortisol differences between poor and rich adults. These authors find evening cortisol concentrations of ~3.7 nmol/l for rich, and ~5.5 nmol/l for poor individuals (where rich and poor are defined as the top and bottom income tertiles). Thus, the difference in cortisol levels between poor and rich individuals is 1.8 nmol/l. It is unlikely that this difference is entirely due to a causal effect of poverty on stress; rather, it is likely to be bidirectional, with poverty leading to stress, but stress also exacerbating poverty. For the sake of simplicity and arugment, let us assume for the moment that 50% of this difference is due to a stress-increasing effect of poverty, i.e. poverty raises evening cortisol levels by 0.9 nmol/l. (Note that it would be not only important to do assess how large this effect truly is, but also relatively easy: a first answer could be gotten from an RCT which exogenously reduces poverty, e.g. through unconditional cash transfers or insurance.)

A typical laboratory stress test induces a cortisol difference of around 10 nmol/l between stress and control groups (e.g. van den Bos, 2009). What are the consequences of such an increase in cortisol levels for economic behavior? Porcelli & Delgado (2009) found that subjects became more risk-averse under laboratory-induced stress. Estimating the change in the risk premium due to stress in their study reveals a jump from 43% without stress to 82% under stress; this corresponds to a 91% increase over baseline levels of the risk premium.

If poverty raises evening cortisol levels by 0.9 nmol/l, and laboratory-induced stress raises them by 10 nmol/l, this suggests that the magnitude of cortisol differences observed between rich and poor is 9% of that induced by a standard laboratory test. The assumption that laboratory and field differences have the same behavioral consequences is both untested and almost certainly overstated; however, at the same time it would be somewhat surprising if the cortisol differences between rich and poor observed in the field had no behavioral consequences at all. If we assume that only a small proportion, say 10%, of the behavioral effects of stress in the lab translate to the field, this predicts that poverty would affect people's risk premia in the field at 0.9% of the effect of stress on risk premia observed in the lab. Put differently, we should expect risk premia among poor people that are about 1% higher than among rich people, as a consequence of higher cortisol levels among this group. This would lower the willingness of poor consumers to invest in risky assets, and thereby lead to reduced growth.

2.10 Conclusion

In the preceding sections, I have summarized evidence suggesting that poverty has particular neurobiological consequences; in particular, it increases cortisol, serotonin, and testosterone levels, and alters the function of these transmitters and hormones in other ways (e.g. it leads to blunted serotonergic responsivity and modulates serotonergic gene-environment interactions). In addition, I have shown that each of these changes, in turn, has profound consequences for economic behavior; altered cortisol, serotonin and testosterone impair decision-making and increase impulsivity. Together, these relationships suggest a neurobiological poverty trap which runs through the channels of cortisol, serotonin, and testosterone.

A number of caveats are in order. First, none of the relationships between poverty and neurobiological outcomes has to date been conclusively shown to be causal, and reverse causality is therefore a serious concern. Future RCTs should make it a priority to measure the effect of poverty alleviation

on levels of these neurobiological markers, and search for natural experiments that allow to assess the effect of poverty increases on them.

Second, it remains to be shown whether any of the mechanisms outlined above are in fact strong enough to perpetuate poverty. Despite the fact that many of the effects described are large in magnitude and solidly established in the literature, the possibility remains that their power is too weak to drive a poverty trap. Data-informed economic models might assess this possibility in the future.

Third, the link between poverty and neurobiological outcomes as I have presented it is somewhat closed-form: the potential effect of poverty on neurobiological variables might be more intuitively conceptualized through psychological variables such as stress, lack of optimism, self-esteem, or depression. The reason to choose neurobiological instead of psychological constructs here was for ease of definition and experimental tractability, even across cultural contexts. However, future studies will have to assess whether focusing on neurobiological rather than psychological variables is sensible.

Finally, I have entirely ignored cultural heterogeneity in the effect of poverty on cortisol, serotonin, and testosterone, and in the effect of these hormones and transmitters on economic behavior. There is no a priori reason to believe that such physiological fundamentals would operate differently across cultures, but the possibility cannot be excluded with complete confidence. Future studies might assess to what extent the neurobiological effects of poverty, and the behavioral effects of neurobiological variables, are culturally specific.

Bibliography

[1] Abeler, J., Falk, A., Goette, L., Huffman, D. (2009). Reference Points and Effort Provision. American Economic Review (in press).

[2] Aggarwal, B.B., Shishodia S., Sandur S.K., Pandey M.K., & Sethi G. (2006). Inflammation and cancer: how hot is the link? Biochemical Pharmacology, 2, 1-17.

[3] Amabile, T.M., Barsade, S.G., Mueller, J.S., & Staw, B.M. (2005). Affect and creativity at work. Administrative Science Quarterly, 50, 367-403.

[4] American Psychiatric Association (1994). Diagnostic and statistical manual of mental health disorders (4th ed). Washington DC: American Psychiatric Publishing.

[5] Argyle, M. (1989). Do happy workers work harder? The effect of job satisfaction on job performance. In: Ruut Veenhoven (Eds), How harmful is happiness? Consequences of enjoying life or not. Rotterdam, Netherlands: Universitaire Pers.

[6] Argyle, M., (2001). The Psychology of Happiness (2nd ed.). London: Routledge.

[7] Arnetz, B.B., Brenner, S.O., Levi, L., Hjelm, R., Petterson, I.L., Wasserman, J., Petrini, B., Eneroth, P., Kallner, A., Kvetnansky, R., et al. (1991). Neuroendocrine and immunologic effects of unemployment and job insecurity. Psychotherapy and Psychosomatics, 55, 76-80.

[8] Ashraf, N., Karlan, D., & Yin, W. (2006). Tying Odysseus to the Mast: Evidence from a Commitment Savings Product in the Philippines. Quarterly Journal of Economics, 121, 635-672.

[9] Banerjee, A. (2007). Making Aid Work: How to fight global poverty – effectively. Boston: Boston Review Books.

[10] Bartels, M., Van den Berg, M., Sluyter, F., Boomsma, D.I., & de Geus, E.J.C. (2003). Heritability of cortisol levels: review and simultaneous analysis of twin studies. Psychoneuroendocrinology 28, 121-137.

[11] Bechara, A., Damasio, A.R., Damasio, H., & Anderson, S.W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. Cognition, 50, 7–15.

[12] Bechara, A., Damasio, H., Damasio, A.R., & Lee, G.P.J. (1999). Dissociation of working memory from decision making within the human prefrontal cortex. Journal of Neuroscience, 19, 5473–5481.

[13] Bennett, A.J., Lesch, K.P., Heils, A., Long, J.C., Lorenz, J.G., Shoaf, S.E., Champoux, M., Suomi, S.J., Linnoila, M.V., & Higley, J.D. (2002). Early experience and serotonin transporter gene variation interact to influence primate CNS function. Molecular Psychiatry, 7, 118–22.

[14] Berger, M., Gray, J.A., & Roth, B.L. (2009). The expanded biology of serotonin. Annual Review of Medicine, 60, 355–66.

[15] Bertrand, M., Karlan, D., Mullainathan, S., Shafir, E., Zinman, J. (2005). What's Pscyhology Worth? A Field Experiment in the Consumer Credit Market. Harvard University Working Paper.

[16] Bertrand, M., Mullainathan, S., & Shafir, E. (2005). A Behavioral Economics View of Poverty. Harvard University Working Paper.

[17] Bolton, G.E., Katok, E., & Zwick, R. (1998). Dictator game giving: Rules of fairness versus acts of kindness. International Journal of Game Theory, 27 , 269-299.

[18] Bolton, P., Neugebauer, R., & Ndogoni, L. (2002). Prevalence of depression in rural Rwanda based on symptom and functional criteria. The Journal of Nervous and Mental Disease, 190, 631-637.

[19] Bolton, P., Wilk, C.M., Ndogoni, L. (2004). Assessment of depression prevalence in rural Uganda using symptom and function criteria. Soc Psychiatry Psychiatr Epidemiol, 39, 442–447.

[20] Brummett, B.H., Boyle, S.H., Kuhn, C.M., Siegler, I.C., & Williams, R.B. (2010). Socioeconomic status moderates associations between CNS serotonin and expression of beta2-integrins CD11b and CD11c. Journal of Psychiatric Research, 44, 373–7.

[21] Camerer, C. (2003). Behavioral Game Theory. Princeton: Princeton University Press.

[22] Chen, E., Cohen, S., & Miller, G.E. (2010). How low socioeconomic status affects 2-year hormonal trajectories in children. Psychological Science, 21(1), 31-37.

[23] Cloninger, C.R. (1987). A systematic method for clinical description and classification of personality variants: a proposal. Archives of General Psychiatry, 44, 573–588.

[24] Coates, J.M., & Herbert, J. (2008). Endogenous steroids and financial risk taking on a London trading floor. Proceedings of the National Academy of Sciences, 105, No. 16, 6167–6172.

[25] Cohen, S., Schwartz, J., et al. (2006a). Socioeconomic status, race and diurnal cortisol levels in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Psychosom Med, 68, 41-50.

[26] Cohen S., Doyle, W.J., & Baum, A. (2006b). Socioeconomic status is associated with stress hormones. Psychosom Med, 68, 414-420.

[27] Coussens, L.M., & Werb, Z. (2002). Inflammation and cancer. Nature, 420, 860-867.

[28] Crisan, L.G., Pana, S., Vulturar, R., Heilman, R.M., Szekely, R., Druga, B., Dragos, N., & Miu, A.C. (2009). Genetic Contributions of the Serotonin Transporter to Social Learning of Fear and Economic Decision Making. Social Cognitive and Affective Neuroscience, 4, 399-408.

[29] Crisp, R. (2007). Depression and occupational disability in five diagnostic groups: a review of recent research. Disabil Rehabil, 29, 267-279.

[30] Crockett, M.J., Clark, L., Tabibnia, G., Lieberman, M.D., & Robbins, T.W. (2008). Serotonin modulates behavioural reactions to unfairness. Science, 320, 1739.

[31] Cutuli, J.J., Wiik, K.L., Herbers, J.E., Gunnar, M.R., & Masten, A.S. (2010). Cortisol function among early school-aged homeless children. Pscyhoneuroendocrinology, 35, 833-845.

[32] da Rocha, F.F., Malloy-Diniz, L., Lage, N.V., Romano-Silva, M.A., de Marco, L.A., & Correa, H. (2008). Decision-making impairment is related to serotonin transporter promoter polymorphism in a sample of patients with obsessive–compulsive disorder. Behavioural Brain Research, 95, 159–63.

[33] Dayan, P., & Huys, Q.J.M. (2009). Serotonin in Affective Control. Annual Review of Neuroscience, 32, 95–126.

[34] de Kloet, E.R., Oitzl, M.S., & Joëls, M (1999). Stress and cognition: are corticosteroids good or bad guys? Trends Neurosci, 22, 422-426.

[35] De Martino, B., Kumaran, D., Seymour, B., & Dolan, R.J. (2006). Frames, biases, and rational decision-making in the human brain. Science, 313, 684–687.

[36] Deakin, J.F.W., & Graeff, F.G. (1991). 5-HT and mechanisms of defence. Journal of Psychopharmacology, 5, 305–315.

[37] Deaton, A.S. (2009). Instruments of development: Randomization in the tropics, and the search for the elusive keys to economic development. NBER Working Paper 14690.

[38] Duflo, E. (2003). Grandmothers and Granddaughters: Old-Age Pensions and Intrahousehold Allocation in South Africa. World Bank Economic Review, 17, 1-25.

[39] Duflo, E., & Kremer, M. (2003). Use of Randomization in the Evaluation of Development Effectiveness. Paper prepared for the World Bank Operations Evaluation Department (OED) Conference on Evaluation and Development Effectiveness in Washington, D.C., 15-16 July.

[40] Duflo, E., Kremer, M., & Robinson, J. (2009). Nudging Farmers to Utilize Fertilizer: Theory and Experimental Evidence from Kenya. MIT Working Paper.

[41] Dulin-Keita, A., Casazza, K., Fernandez, J.R., Goran, M.I., & Gower, B. (2010). Do neighborhoods matter? Neighborhood disorder and long-term trends in serum cortisol levels. J Epidemiol Community Health (in press).

[42] Dupas, P. (2005). Relative Risks and the Market for Sex: Teenagers, Sugar Daddies and HIV in Kenya. Working Paper.

[43] Easterlin, R.A. (1974). Does Economic Growth Improve the Human Lot? In David, P.A., & Reder, M. W. (Eds.), Nations and Households in Economic Growth: Essays in Honor of Moses Abramovitz. New York: Academic Press, Inc.

[44] Easterly, W. (2006). The White Man's Burden: Why the West's Efforts to Aid the Rest Have Done So Much Ill and So Little Good. New York: Penguin.

[45] Easterly, W. (2009). Can the West Save Africa? Journal of Economic Literature, 47, 373-447.

[46] Evans, G.W., & English, K. (2002). The environment of poverty: multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. Child Development, 2002, 73, 1238–1248.

[47] Evans, G.W., & Schamberg, M.A. (2009). Childhood poverty, chronic stress, and adult working memory. Proc Natl Acad Sci U S A, 106, 6545-6549.

[48] Evans, G.W., & Kim, P. (2009). Childhood poverty and health: cumulative risk exposure and stress dysregulation. Psychological Science, 2007, 18, 953–57.

[49] Fernald, L.C.H., & Gunnar, M.R. (2009). Poverty-alleviation program participation and salivary cortisol in very low-income children. Social Science & Medicine, 68, 2180-2189.

[50] Fernald, L.C.H., Hamad, R., Karlan, D., Ozer, E., & Zinman, J. (2008). Small individual loans and mental health: a randomized controlled trial among South African adults. BioMed Central Public Health, 8, 409.

[51] Ferracuti, S., Seri, S., Mattia, D., & Cruccu, G. (1994). Quantitative EEG modifications during the cold water pressor test:

Hemispheric and hand differences. International Journal of Psychophysiology, 17, 261-268.

[52] Fournier, J.C.; DeRubeis, R.J.; Hollon, S.D.; Dimidjian, S.; Amsterdam, J.D.; Shelton, R.C.; Fawcett, J. (2010). Antidepressant drug effects and depression severity. The Journal of the American Medical Association, 303, 47–53.

[53] Friston, K.J., Buechel, C., Fink, G.R., Morris, J., Rolls, E., & Dolan, R.J. (1997). Psychophysiological and modulatory interactions in neuroimaging. Neuroimage, 6, 218 –229.

[54] Garcia-Alvarez, R. (1986). Epidemiology of depression in Latin America. Psychopathology, 19, 22–25.

[55] Gray, J. (1982). The Neuropsychology of Anxiety. London: Oxford University Press.

[56] Gray, J. R. (1999). A bias toward short-term thinking in threat-related negative emotional states. Personality and Social Psychology Bulletin, 25, 65–75.

[57] Greenberg, B.D., Li, Q., Lucas, F.R., Hu S., Sirota, L.A., Benjamin, J., Lesch, K.P., Hamer, D., & Murphy, D.L. (2000). Association between the serotonin transporter promoter polymorphism and personality traits in a primarily female population sample. American Journal of Medical Genetics, 96, 202–216.

[58] Ha, R.Y., Namkoong K., Kang, J.I., Kim, Y.T., & Kim, S.J. (2009). Interaction between serotonin transporter promoter and dopamine receptor D4 polymorphisms on decision making. Progress in Neuro-Psychopharmacology & Biological Psychiatry, 33, 1217–1222.

[59] Hagerty, M.R., & Veenhoven, R. (2003). Wealth and happiness revisited: Growing wealth of nations does go with greater happiness. Social Indicators Research, 64, 1-27.

[60] Hammen, C. (2005). Stress and Depression. Annual Review of Clinical Psychology, 1, 293-319.

[61] Hansenne, M., & Ansseu, M. (1999). Harm avoidance and serotonin. Biological Psychology, 51,77–81.

[62] Herrnstein, R. J., Prelec, D., & Vaughan, W., Jr. (1986). An intrapersonal prisoner.s dilemma. Paper presented at the 19th Symposium on the Quantitative Analysis of Behavior, Harvard University, Cambridge, MA.

[63] Higley, J.D., Suomi, S.J., & Linnoila, M. (1992). A longitudinal assessment of CSF monoamine metabolite and plasma cortisol concentrations in young rhesus monkeys. Biological Psychiatry, 32, 127– 45.

[64] Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. Neuropsychopharmacology, 23, 477-501.

[65] Homberg, J.R., van den Bos, R., den Heijer, E., Suer, R., & Cuppen, E. (2008). Serotonin transporter dosage modulates long-term decision-making in rat and human. Neuropharmacology, 55, 80–4.

[66] Imbens, G. W. (2009). Better LATE than Nothing: Some Comments on Beaton (2009) and Heckman and Urzua (2009). Harvard University Working Paper.

[67] Jokela, M., Lehtima, T., & Keltikangas-Järvinen, L. (2007). The Serotonin Receptor 2A Gene Moderates the Influence of Parental Socioeconomic Status on Adulthood Harm Avoidance. Behavior Genetics, 37, 567–574.

[68] Jollant, F., Buresi, C., Guillaume, S., Jaussent, I., Bellivier, F., Leboyer, M., Castelnau, D., Malafosse, A., & Courtet P. (2007). The influence of four serotonin-related genes on decision-making in suicide attempters. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 144B, 615–24.

[69] Keinan, G. (1987). Decision making under stress: Scanning of alternatives under controllable and uncontrollable threats. Journal of Personality and Social Psychology, 52, 639–644.

[70] Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. Proceedings of the National Academy of Sciences of the United States of America, 100, 9090-9095.

[71] Kim, J.J., & Haller, J. (2007). Glucocorticoid hyper- and hypofunction: stress effects on cognition and aggression. Ann N Y Acad Sci, 1113, 291-303.

[72] Kirschbaum, C., Kudielka, B.M., Gaab, J., Schommer, N.C., & Hellhamer, D.H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary- adrenal axis.

[73] Kirschbaum, C., Pirke, K.M., & Hellhamer, D.H., (1993). The 'Trier Social Stress Test' — a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology, 28, 76-81.

[74] Klein, G. (1996). The effect of acute stressors on decision making. In J. E. Driskell & E. Salas (Eds.), Stress and human performance (pp. 49–88). Mahwah, NJ: Erlbaum.

[75] Kraft, A.J., & Luecken, L.J. (2009). Childhood parental divorce and cortisol in young adulthood: evidence for mediation by family income. Psychoneuroendocrinology, 34, 1363-1369.

[76] Kremer, M., Miguel, M., Mullainathan, S., Null, C., & Zwane, A. P. (2009). Making Water Safe: Price, Persuasion, Peers, Promoters, or Product Design? Harvard University Working Paper.

[77] Kuhnen, C.M., & Chiao, J.Y. (2009). Genetic determinants of financial risk taking. Public Library of Science One, 4, e4362.

[78] Laibson, D. (1997). Golden Eggs and Hyperbolic Discounting. Quarterly Journal of Economics, 112, 443-477.

[79] Lawrence, E. C. (1991). Poverty and the Rate of Time Preference: Evidence from Panel Data. Journal of Political Economy, 99, 54-77.

[80] Lerner, D., & Henke, R.M. (2008). What does research tell us about depression, job performance, and work productivity? J Occup Environ Med, 50, 401-410.

[81] Lesch, K.P., Bengel, D., Heils, A., Sabol, S.Z., Greenberg, B.D., Petri, S., Benjamin, J., Muller, C.R., Hamer, D.H., & Murphy, D.L. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. Science, 274, 1527-31.

[82] Li, L., Power, C., Kelly, S., Kirschbaum, C., & Hertzman, C. Life-time socio-economic position and cortisol patterns in mid-life. Psychoneuroendocrinology, 2007, 32, 824–33.

[83] Long, A.B., Kuhn, C.M., & Platt, M.L. (2009). Serotonin shapes risky decision making in Monkeys. Social Cognitive and Affective Neuroscience, 4, 346–356.

[84] Lupien S.J., King, S., Meaney, M.J., & McEwen, B.S. (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. Development and Psychopathology,13, 653–76.

[85] Lupien, S.J., King, S., Meaney, M.J., & McEwen, B.S. (2000). Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. Biological Psychiatry, 48, 976–80.

[86] Lupien, S.J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T.E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. Brain Cogn, 65, 209-237.

[87] Lupien, S.J., McEwen, B.S., Gunnar, M.R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci, 10, 434-445.

[88] Lynch, J. W., Kaplan, G. A., & Shema, S. J. (1997). Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. N Engl J Med, 337, 1889-1895.

[89] Manuck, S.B., Bleil, M.E., Petersen, K.L., Flory, J.D., Mann, J.J., Ferrell, R.E., & Muldoon, M.F. (2005). The socio-economic status of communities predicts variation in brain serotonergic responsivity. Psychological Medicine, 35, 519–28.

[90] Manuck, S.B., Flory, J.D., Ferrell, R.E., & Muldoon, M.F. (2004). Socio-economic status covaries with central nervous system serotonergic responsivity as a function of allelic variation in the serotonin transporter gene-linked polymorphic region. Psychoneuroendocrinology, 29, 651–68.

[91] Manuck, S.B., Flory, J.D., McCaffery, J.M., Matthews, K.A., Mann, J.J., & Muldoon, M.F. (1998). Aggression, impulsivity, and central nervous system serotonergic responsivity in a non-patient sample. Neuropsychopharmacology, 19, 287–99.

[92] Maqsud, M., & Rouhani, S. (1991). Relationships between socioeconomic status, locus of control, self-concept, and academic achievement of batswana adolescents. Journal of Youth and Adolescence, 20, 107-114.

[93] Matthews, K.A., Flory, J.D., Muldoon, M.F., & Manuck, S.B. (2000). Does socioeconomic status relate to central serotonergic responsivity in healthy adults? Psychosomatic Medicine, 62, 231–7.

[94] Mazur, J. E. (1987). An adjusting procedure for studying delayed reinforcement. In M. L. Commons, J. E. Mazur, J. A. Nevin & H. Rachlin (Eds.), Quantitative analyses of behavior: V. The effect of delay and of intervening events on reinforcement value (pp. 55-73). Hillsdale, NJ: Lawrence Erlbaum.

[95] McEwen, B. S. (2004). Protection and damage from acute and chronic stress: allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. Annals of the New York Academy of Sciences, 1032, 1-7.

[96] McEwen, B. S., & Sapolsky, R. M. (1995). Stress and cognitive function. Current Opinion in Neurobiology, 5, 205-216.

[97] McEwen, B.S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. Annals of the New York Academy of Sciences, 896, 30–47.

[98] Miguel, E., & Kremer, M. (2004). Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities. Econometrica 72, 159-217.

[99] Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. Science, 244, 933-938.

[100] Moyo, D. (2009). Dead Aid: Why Aid Is Not Working and How There is Another Way for Africa. New York: Farrar, Straus and Giroux.

[101] Munafo, M.R., Brown, S.M., & Hariri, A.R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: a meta-analysis. Biological Psychiatry, 63, 852–857.

[102] Murphy, S.E., Longhitano, C., Ayres, R.E., Cowen, P.J., Harmer, C.J., & Rogers, R.D. (2009). The role of serotonin in nonnormative risky choice: The effects of tryptophan supplements on loss aversion in healthy adult volunteers. Journal of Cognitive Neuroscience, 21, 9, 1709–1719.

[103] Must, A., Juhasz, A., Rimanoczy, A., Szabo, Z., Keri, S., & Janka, Z. (2007). Major depressive disorder, serotonin transporter, and personality traits: why patients use suboptimal decision-making strategies? Journal of Affective Disorders, 103, 273–6.

[104] Nobile, M., Giorda, R., Marino, C., Carlet, O., Pastore, V., Vanzin, L., Bellina, M., Molteni, M., & Battaglia, M. (2007). Socioeconomic status mediates the genetic contribution of the dopamine receptor D4 and serotonin transporter linked promoter region repeat polymorphisms to externalization in preadolescence. Development and Psychopathology, 19, 1147–60.

[105] Noble, K. G., Norman, M. F., & Farah, M. J. (2007). Socioeconomic gradients predict individual differences in neurocognitive abilities. Developmental Science, 10, 464-80.

[106] Noble, K. G., Norman, M. F., Farah, M. J. (2005). Neurocognitive correlates of socioeconomic status in kindergarten cildren. Developmental Science, 8, 74–87.

[107] Oswald, A.J. (2009). Happiness and Productivity. University of Warwick Working Paper.

[108] Ouellet-Morin, I., Dionne, G., Pérusse, D., Lupien, S.J., Arseneault, L., Barr, R.G., Tremblay, R.E., Boivin, M. (2009). Daytime cortisol secretion in 6-month-old twins: genetic and environmental contributions as a function of early familial adversity. Biol Psychiatry, 65, 409-416.

[109] Patel, V., Araya, R., & Bolton, P. (2004). Editorial: Treating depression in the developing world. Tropical Medicine & International Health, 9, 539.

[110] Patel, V., Chisholm, D., Rabe-Hesketh, S., Dias-Saxena, F., Andrew, G., & Mann, A. (2003). Efficacy and cost-effectiveness of drug and psychological treatments for common mental disorders in general health care in Goa, India: a randomised, controlled trial. Lancet, 361, 33-39.

[111] Paxson, C., & Schady, N. (2007). Does money matter ? The effects of cash transfers on child health and development in rural Ecuador. Policy Research Working Paper Series 4226, The World Bank, Washington, D.C.

[112] Peirson, A.R., Heuchert, J.W., Thomala, L., Berk, M., Plein, H., & Cloninger, C.R. (1999). Relationship between serotonin and the temperament and character inventory. Journal of Psychiatric Research, 89, 29–37.

[113] Polesskaya, O.O., & Sokolov, B.P. (2002). Differential expression of the 'C' and 'T' alleles of the 5-HT2A receptor gene in the temporal cortex of normal individuals and schizophrenics. Journal of Neuroscience Research, 67, 812–822.

[114] Porcelli, A. J., & Delgado, M. R. (2009). Acute stress modulates risk taking in financial decision making. Psychological Science, 20, 278-283.

[115] Preston, S. D., Buchanan, T. W., Stansfield, R. B., & Bechara. A. (2007). Effects of Anticipatory Stress on Decision Making in a Gambling Task. Behavioral Neuroscience, 121, No. 2, 257–263.

[116] Puttonen, S., Ravaja, N., Keltikangas-Ja¨rvinen, L. (2005). Cloninger's temperament dimensions and affective responses to different challenges. Comprehensive Psychiatry, 46, 128–134.

[117] Quattrone, A., Tedeschi, G., Aguglia, U., Scopacasa, F., Direnzo, G.F., & Annunziato, L. (1983). Prolactin secretion in man: a useful tool to evaluate the activity of drugs on central 5-hydroxytryptaminergic neurones. Studies with fenfluramine. British Journal of Clinical Pharmacology, 16, 471–5.

[118] Reilly, J.G., McTavish, S.F., Young, A.H. (1997). Rapid depletion of plasma tryptophan: a review of studies and experimental methodology. Journal of Psychopharmacology, 11, 381–92.

[119] Robb, K. A., Simon, A. E., & Wardle, J. (2009). Socioeconomic Disparities in Optimism and Pessimism. Internation Journal of Behavioral Medicine, 16, 331-338.

[120] Robbins, T.W., & Crockett, M.J. (2010. Role of Central Serotonin in Impulsivity and Compulsivity: Comparative Studies in Experimental Animals and Humans. In: Christian Müller & Barry Jacobs (Eds.), Handbook of the Behavioral Neurobiology of Serotonin. Oxford: Elsevier Academic Press.

[121] Rogers, R.D., Everitt, B.J., Baldacchino, A., Blackshaw, A.J., Swainson, R., Wynne, K., Baker, N.B., Hunter, J., Carthy, T., Booker, E., London, M., Deakin, J.F.W., Sahakian, B.J. & Robbins, T.W. (1999). Dissociable deficits in the decision- making cognition of chronic amphetamine abusers, opiate abusers, patients with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: evidence for monoaminergic mechanisms. Neuropsychopharmacology, 20, 322–339.

[122] Rogers, R.D., Tunbridge, E.M., Bhagwagar, Z., Drevets, W.C., Sahakian, B.J., & Carter, C.S. (2003). Tryptophan depletion alters the decision-making of healthy volunteers through altered processing of reward cues. Neuropsychopharmacology, 28, 153–62.

[123] Roiser, J.P., de Martino, B., Tan, G.C., Kumaran, D., Seymour, B., Wood, N.W., & Dolan, R.J. (2009). A genetically mediated bias in decision making driven by failure of amygdale control. Journal of Neuroscience Methods, 29, 5985–91.

[124] Ross, R. (1999). Atherosclerosis - An inflammatory disease. New England Journal of Medicine, 340, 115-126.

[125] Sachs, J. (2005). The End of Poverty: Economic Possibilities for Our Time. New York: Penguin.

[126] Sadeh, N., Javdani, S., Jackson, J.J., Reynolds, E.K., Potenza, M.N., Gelernter, J., Lejuez, C.W., & Verona, E. (2010). Serotonin Transporter Gene Associations With Psychopathic Traits in Youth Vary as a Function of Socioeconomic Resources. Journal of Abnormal Psychology, 119, No. 3, 604–609.

[127] Saridjan, N.S., Huizink, A.C., Koetsier, J.A., Jaddoe, V.W., Mackenbach, J.P., Hofman, A., Kirschbaum, C., Verhulst, F.C.,

& Tiemeier, H. (2010). Do social disadvantage and early family adversity affect the diurnal cortisol rhythm in infants? The Generation R Study. Hormones and Behaviour, 57, 247-54.

[128] Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: assessment and implications of generalized outcome expectancies. Health Psychol, 4, 219-247.

[129] Schildkraut, J.J. (1965). The catecholamine hypothesis of affective disorders: A review of supporting evidence. Am J Psychiatry, 122, 509-522.

[130] Sherman, L., & Hofmann, R. J. (1978). Interrelationship amongst locus of control, socioeconomic status, sex and achievment. Paper presented at the Annual Convention of the American Psycological Association. Toronto, Ontario, Canada.

[131] Sobocki, P., Jönsson, B., Angst, J., & Rehnberg, C. (2006). Cost of depression in Europe. J Ment Health Policy Econ, 8, 87-98.

[132] Soubrie, P. (1986). Reconciling the role of serotonin neurons in human and animal behaviour. Behavioral and Brain Sciences, 9, 319–364.

[133] Spears, D. (2011). Economic decision-making in poverty depletes behavioral control. CEPS Working Paper No. 213, Princeton University.

[134] Steptoe, A., Owen, N., Kunz-Ebrecht, S., & Mohamed-Ali, V. (2002). Inflammatory cytokines, socioeconomic status, and acute stress responsivity. Brain, Behavior, and Immunity, 16, 774-784.

[135] Steptoe, A., Willemsen, G., Owen, N., Flower, L., & Mohamed-Ali, V. (2001). Acute mental stress elicits delayed increases in circulating in_ammatory cytokine levels. Clinical Science (London, England: 1979), 101, 185-192.

[136] Stevenson, B., & Wolfers, J. (2008). Economic Growth and Subjective Well-Being: Reassessing the Easterlin Paradox. Brookings Papers on Economic Activity.

[137] Straub, R.H., (2004). Complexity of the bi-directional neuroimmune junction in the spleen. Trends in Pharmacological Sciences, 25, 640-646.

[138] Takahashi, T., Ikeda, K., Ishikawa, M., Kitamura, N., Tsukasaki, T., Nakama, D., & Kameda, T. (2005). Interpersonal trust and social stress-induced cortisol elevation. Neuroreport , 16 No. 28, 197-199.

[139] Talbot, P.S., Watson, D.R., Barrett, S.L., & Cooper, S.J. (2006). Rapid Tryptophan Depletion Improves Decision-Making Cognition in Healthy Humans without Affecting Reversal Learning or Set Shifting. Neuropsychopharmacology, 31, 1519–1525.

[140] Tanaka, T., Camerer, C.F., & Nguyen, Q. (2010). Risk and Time Preferences: Linking Experimental and Household Survey Data from Vietnam. American Economic Review, 100, 557–571.

[141] Taylor, S. E., & Seeman, T. E. (1999). Psycosocial resources and SES-health relationship. Ann NY Acad Sci, 896, 210-225.

[142] Thaler, R.H., & Benartzi, S. (2007). Save More Tomorrow: Using Behavioral Economics to Increase Employee Saving. Journal of Political Economy, 112, 164-187.

[143] Thavichachart, N., Intoh, P., Thavichachart, T., Meksupa, O., Tangwongchai, S., Sughondhabirom, A., Worakul, P. (2001) Epidemiological survey of mental disorders and knowledge attitude practice upon mental health among people in Bangkok Metropolis. J Med Ass Thailand, 84, 118–126.

[144] Turecki, G., Brie're, R., Dewar, K., Antonetti, T., Lesage, A.D., Se´guin, M., Chawky, N., Vanier, C., Alda, M., Joober, R., Benkelfat, C., & Rouleau, G.A. (1999). Prediction of level of serotonin 2A receptor binding by serotonin receptor 2A genetic variation in postmortem brain samples from subjects who did or did not commit suicide. American Journal of Psychiatry, 156, 1456–1458.

[145] Twenge, J. M., & Campbell, K. W. (2002). Self-Esteem and Socioeconomic Status: A Meta-Analytic Review. Personality and social psychology review, 6, 59-71.

[146] UN (2007). Millennium Development Report.

[147] UNDP (2009). Human Development Report.

[148] UNICEF (2000). Progress of Nations Report.

[149] van den Bos, R., Harteveld, M., & Stoop, H. (2009). Stress and decision-making in humans: Performance is related to cortisol reactivity, albeit differently in men and women. Psychoneuroendocrinology, 34, 1449-1458.

[150] van Heeringen, C., Audenaert, K., Van Laere, K., Dumont, F., Slegers, G., Mertens, J., & Dierckx, R.A. (2003). Prefrontal 5-HT2a receptor binding index, hopelessness and personality characteristics in attempted suicide. Journal of Affective Disorders, 74, 149–158, 574.

[151] Williams, R.B., Marchuk, D.A., Siegler, I.C., Barefoot, J.C., Helms, M.J., Brummett, B.H., Surwit, R.S., Lane, J.D., Kuhn, C.M., Gadde, K.M., Ashley-Koch, A., Svenson, I.K., & Schanberg, S.M. (2008). Childhood Socioeconomic Status and Serotonin Transporter Gene Polymorphism Enhance Cardiovascular Reactivity to Mental Stress. Psychosomatic Medicine, 70, 32–39.

[152] Wilckens T. 1995 Glucocorticoid and immune function: physiological relevance and pathogenic potential of hormonal dysfunction. Trends Pharmacol Sci. 16:193–197.

[153] Wise, C.D., Stein, L. & Berger, B.D. (1972). Benzodiazepines – reducing anxiety by reduction of serotonin turnover in brain. Science, 177, 180–183.

[154] World Bank (2001). World Development Report (WDR) 2001: Attacking Poverty.

[155] World Bank (2003). World Development Report (WDR) 2003: Investing in Health.

[156] World Health Organization (2001). World Health Report (WHR) 2001: Mental Health: New Understanding, New Hope.

[157] Yatham, L.N., & Steiner, M. (1993). Neuroendocrine probes of serotonergic function: a critical review. Life Sciences, 53, 447–63.

Chapter 3

Low Income is Associated with High Baseline Levels and Low Stress Reactivity of Cortisol, but Not Alpha Amylase

3.1 Summary

Does poverty have particular neurobiological consequences? We test here whether low-income individuals show higher baseline levels, or higher stress-induced reactivity, of the stress hormone cortisol and the sympathetic marker salivary alpha-amylase. A sample of 80 healthy university students was sampled both at baseline, and while performing the Trier Social Stress Test (TSST), a standard stress-inducing laboratory task. We find that poor individuals have higher baseline levels of cortisol, and lower cortisol reactivity to stress, than richer individuals. In contrast, salivary alpha amylase shows no relationship to income, neither in baseline levels nor stress reactivity. Together, these findings suggest that poverty is associated with dysregulation of cortisol; this may lead to adverse health outcomes and disadvantageous decision-making.

3.2 Introduction

Does poverty get under your skin? A prominent hypothesis is that low-income environments may be characterized by both greater exposure to stressful events, and the absence of resources to deal with such stress (Baum et al., 1999; Steptoe et al., 2002; Brunner, 1997; Kristenson, 2004). This

hypothesis predicts changes in physiological markers of stress: if low-income populations are more stressed, they should show higher levels, altered daily profiles, or different stress reactivity in stress hormones. In the following, we refer to this claim as the poverty-stress hypothesis. These changes could in turn lead to adverse consequences of their own, such as immunosuppression (Cacioppo et al., 2002) or altered decision-making (Porcelli & Delgado, 2009). Do low-income populations indeed show differences in physiological markers of stress? A growing body of literature investigates this question; however, it will emerge in the following discussion that the answer remains unclear.

The physiological response to stress in humans is characterized by the joint activation of two systems: the hypothalamic-adrenal-pituitary (HPA) axis and its associated stress hormone cortisol, and the sympathetic-adrenal-medullary (SAM) axis and its associated stress hormone norepinephrine (NE). Cortisol can be measured directly in saliva, where it is a good indicator of levels in the blood (Kirschbaum & Hellhammer, 1994); sympathetic can be measured by proxy in saliva, through salivary alpha-amylase (sAA; van Stegeren et al., 2006; Ehlert et al., 2006).

Most attention in the literature has been devoted to cortisol. A number of studies show higher baseline levels of cortisol in populations of low socioeconomic status (SES), consistent with the poverty-stress hypothesis (Cohen et al., 2006a, 2006b; Evans & Kim, 2009; Evans & English, 2002; Li et al., 2007; Lupien et al., 2000; Arnetz et al., 1991; see Dowd et al., 2009, for a review). However, other studies show no associations, or relationships restricted to certain population groups or parts of the daily cortisol profile (Dowd et al., 2006; Gersten, 2008; Goodman et al., 2005; Ranjit et al., 2005; Rosmond & Bjorntorp, 2000; Steptoe et al., 2005; Decker, 2000; Rosero-Bixby & Dow, 2009). Finally, three studies even show a positive association between baseline cortisol and SES (Brandstadter et al., 1991; Chen & Paterson, 2006; Fiocco et al., 2007). Thus, it remains unclear to what extent low SES is in fact associated with higher baseline cortisol levels; the first purpose of the present study was to shed further light on this question.

Second, a question that has received much less attention is whether cortisol reactivity to stress is altered in people with low SES. In the few studies that have addressed this question, participants are typically presented with mildly stressful tasks in the laboratory, such as the well-known Trier Social Stress Task (TSST; Kirschbaum et al., 1993). The results are conflicting: Fiocco et al. (2007) find a negative association between cortisol reactivity and SES, Kristenson et al. (2001) find a positive association, Adler et al. (2000) no association between reactivity and SES, but one between SES and adaptation to repeated stress; and two other studies find no relationship

(Steptoe et al., 2005; Kapuku et al., 2002). Thus, the second aim of this paper was to investigate further to what extent cortisol reactivity to stress is associated with income.

The literature on SES and sAA is smaller yet; two studies show higher sAA levels in low-SES children (Wolf et al., 2008; Granger et al., 2006), but one of these only obtained an effect in asthmatic and not healthy children (Wolf et al., 2008), and no study has investigated this link in adults. In addition, to our knowledge no study has investigated whether sAA reactivity to stress is modulated by income. Thus, the final goals of this paper were to investigate whether baseline levels of sAA, and sAA reactivity to stress, are affected by income in adults.

To address these research questions, we measured both baseline levels of cortisol and sAA, and cortisol and sAA reactivity to a laboratory stressor, in a sample of Swiss university students. We find that lower income students have higher levels of baseline cortisol, and reduced cortisol reactivity to stress, compared to richer students. However, no relationships between income and baseline sAA levels were found; furthermore, income did not predict sAA reactivity to laboratory stress.

3.3 Methods

3.3.1 Participants

We recruited 81 healthy male participants form the subject pool of the University of Zürich. Their mean age was 21.31 $\pm$ 1.85 years. We excluded students of economics and psychology, and those who were acutely or chronically ill, took medications, drugs, smoked more than 5 cigarettes a day, regularly consumed more than 60g of alcohol per day, suffered from allergies or psychiatric disorders, were in psychological or psychiatric treatment at the time of the study, had previously participated in a TSST, or had a body mass index smaller than 18 or greater than 25. Participants were instructed to not consume medications, alcohol, or coffee, and not to engage in sexual intercourse, for 24h before the experiment. In addition, they were asked to get up at least 3h before the beginning of the experiment, and to not drink coffee, eat, smoke, or perform strenuous physical activity in the last 2h before the experiment. All participants were tested in the afternoon between 2pm and 8pm, when plasma cortisol levels are close to the circadian trough. They gave written informed consent and were reimbursed for their participation. An experimental session lasted 2h.

3.3.2 Stress Manipulation

Psychosocial stress was induced with a grouped version of the Trier Social Stress Test (TSST-G; Kirschbaum et al., 1993; von Dawans et al., 2010). The procedure followed closely that described by Dawans et al. (2010), and involved a preparation period of 5 min, followed by a video- and audio- taped public speaking task of 12 min (a fictional job interview, see below), and a mental arithmetic task of 8 min, both in front of an evaluation committee (one man and one woman wearing white laboratory coats). A maximum of 4 and a minimum of 2 subjects were tested at the same time. In the job interview component of the task, each participant had 3 minutes to describe why their personal qualities qualified them for a job. The committee repeatedly interrupted the presentation with questions, following a pre-prepared script. In the arithmetic task, participants were asked to count backwards in steps of 16, starting at a random 4-digit number. Mistakes were corrected by the panel, and the participant had to start over. All Subjects first delivered their speech and after that performed the arithmetic task. Each subject was called at least twice and in random order for every task, to induce a feeling of unpredictability. Speaking time for every participant was kept constant.

To keep the cognitive load and circumstances of the control condition as comparable as possible, only lacking the component of social control, subjects in the control condition underwent the same conditions, with three important differences. First, subjects were not video- or audio-taped and there was no panel in laboratory coats, just a passive observer in a corner of the room. Second, while the mental arithmetic task was the same, the fictional job interview was replaced by an account of a memorable experience with a good friend. The purpose of this task was toe require a similar amount of creativity and cognitive resources as the job interview, while not containing the same stressful element of social evaluation and having to "talk oneself up". Finally, all subjects performed their tasks simultaneously with the other participants; this made the individual contributions unintelligible to the passive observer and the other participants, thus further reducing the social evaluative element. Total duration of the task and speaking time for each participant were matched to the parameters of the stress condition.

3.3.3 Procedure

Subjects were randomly assigned to one of two conditions: control (N=41) and stress (N=40). Subjects were instructed not to talk to each other during the whole experiment. An overview of the study timeline is displayed in Figure 3.4.1. Twenty minutes after subjects arrived in the laboratory, a

first saliva sample was taken. Subjects were guided to a room where they received general instructions about the experiment, but not the TSST or Control tasks, to avoid inducing anticipatory stress before taking the second baseline saliva sample. After 20 min, second saliva sample was taken. Next, subjects received instructions for the TSST, and were given a 5 min preparation period for the stress or control task. They were then guided to another room, where they gave their speech. Before subjects were instructed to perform the arithmetic task, a third saliva sample was taken. Directly after the whole TSST or control procedure, a fourth saliva sample was taken. Next, participants were asked to sit at the chair placed behind them and performed economic choice tasks, the results of which are reported in a separate paper. After completing these tasks, participants remained seated, filled in a socioeconomic questionnaire, and read a neutral magazine. Additional saliva samples were taken 10 min, 20 min, and 50 min after the end of the TSST or control task. After the last saliva sample was taken, participants were debriefed and paid for their participation.

3.3.4 Salivary Sampling and Biochemical Analysis

Salivary samples were obtained using Salivette sampling devices (Sarstedt, Nümbrecht, Germany) at 7 time points during the experiments (Figure 3.4.1). Salivary samples were stored at -20°C until further analysis. Free cortisol levels were measured using a commercially available immunoassay (IBL, Hamburg, Germany). Salivary alpha-amylase levels were measured by a quantitative enzyme kinetic essay as described elsewhere (van Stegeren et al., 2006).

3.3.5 Income measure

Information about the individual incomes of participants was elicited with the following question: "How much money do you have available in an average month (excluding costs of rent and insurance)?" The purpose of this particular formulation was to elicit income data that was reflective of the financial liquidity of participants and independent from the source of the money, e.g. jobs vs. parents. Statistical analysis was performed on both the untransformed and log-transformed income data (see below).

3.3.6 Covariates

Our regressions control for the following covariates: age, measured in years; time between waking and the beginning of the experimental session (timeawake);

body mass index (bmi), defined as weight (kg) / height2 (cm); number of siblings (numsibs); political orientation (left = 0, right = 100; polright); and dummy variables for whether subjects had recently smoked (recentsmoke), eaten (recenteat), or drunk coffee (recentcoffee) or alcohol (recentalcohol). To show the robustness of the effects, we report different specifications controlling for various subsets of these covariates.

3.3.7 Statistical Analysis

Baseline cortisol and sAA were determined by averaging the cortisol and sAA levels from the first two of the seven saliva samples. Stress responsivity of cortisol and sAA was defined as the area under the cortisol or sAA response curve (AUC) in those subjects exposed to the social stressor (stress group). The AUC was calculated with respect to the baseline to rule out effects stemming from baseline differences. The effectiveness of the stress task in raising hormone levels was assessed using a 7 (Sample Period: t0 vs. t20 vs. t30 vs. t40 vs. t50 vs. t60 vs. t90) x 2 (Stress: TSST-G vs. Control) General Linear Model (GLM) repeated measures ANOVA with Sample Period as a repeated measure.

The relationship between baseline hormone levels or hormone stress responsivity and income was assessed with ordinary least squares regression using heteroskedasticity-robust standard errors. Specifically, we regressed hormone levels (either baseline or AUC) on income (either linear or log), and varying sets of covariates as described above. When assessing stress reactivity, the regressions were restricted to those subjects who were exposed to the TSST. Different specifications include different subsets of covariates to show the robustness of our results.

3.4 Results

3.4.1 Effectiveness of TSST in raising cortisol and sAA levels

The experimental groups (stress vs. control) did not differ in age, BMI, or baseline cortisol and sAA (P's > 0.1). As expected, the stress manipulation significantly raised both cortisol and sAA levels: an ANOVA for cortisol showed a significant Sample Period x Stress interaction (Figure 3.4.1A, $F_{1.6,124.4}=34.70$, $P<0.001$). Furthermore, a main effect of Sample Period ($F_{1.6,124.4}=34.02$, $P<0.001$) and a main effect of Stress ($F_{1,77}=38.53$, $P<0.001$) were found. Planned simple contrasts related to baseline showed that sub-

jects in the stress condition had increased cortisol levels from t30 (during the TSST-G) until t90, i.e. at the end of the session (all P's$<$0.001).

For alpha-amylase, a significant Sample Period x Stress interaction (Figure 3.4.1B, $F_{5.1,395}$=8.89, P$<$0.001) and a significant main effect of Sample Period ($F_{5.1,395}$=23.12, P$<$0.001) were found. Planned simple contrasts compared to baseline showed that alpha-amylase levels were increased in the stress condition from t30, i.e. during the TSST-G, until after the early test condition at t50 (P's$<$0.05).

3.4.2 Cortisol and income

Our first main question was whether baseline cortisol is associated with income: do poorer people have higher cortisol levels? To answer this question, we regressed baseline cortisol levels on income, while controlling for various subsets of covariates, and using both linear and log specifications for the income variable. The results are shown in Table 3.4.1 (linear income variable) and Table 3.4.2 (log income variable). The income coefficient is significant in all specifications, with higher incomes associated with lower baseline cortisol levels, and low-income subjects showing higher baseline cortisol levels.

Second, we wished to test whether cortisol reactivity to the TSST was associated with income: do poorer people have higher or lower stress reactivity? To answer this question, we regressed cortisol AUC on income, again using both linear and log specifications, and controlling for various covariates. The results are shown in Table 3.4.3 (linear income variable) and Table 3.4.4 (log income variable). The coefficient on income is significantly positive in all specifications except that which does not control for the time elapsed between waking and testing, or any other covariates. Thus, higher-income subjects show higher stress reactivity in terms of cortisol AUC than lower-income participants.

3.4.3 sAA and income

Our third question was whether baseline sAA is associated with income. We proceeded as above for the relationship between baseline cortisol and income. The results are presented in Table 3.4.5 (linear income variable) and Table 3.4.6 (log income variable). None of the specifications resulted in a significant coefficient on income. Thus, baseline sAA does not appear to be related to income, whether it is specified linearly or as log.

Finally, we asked whether sAA reactivity to the TSST was associated with income. Again we proceeded as above for cortisol, using sAA AUC as the dependent variable. The results are shown in Tables 3.4.7 and 3.4.8.

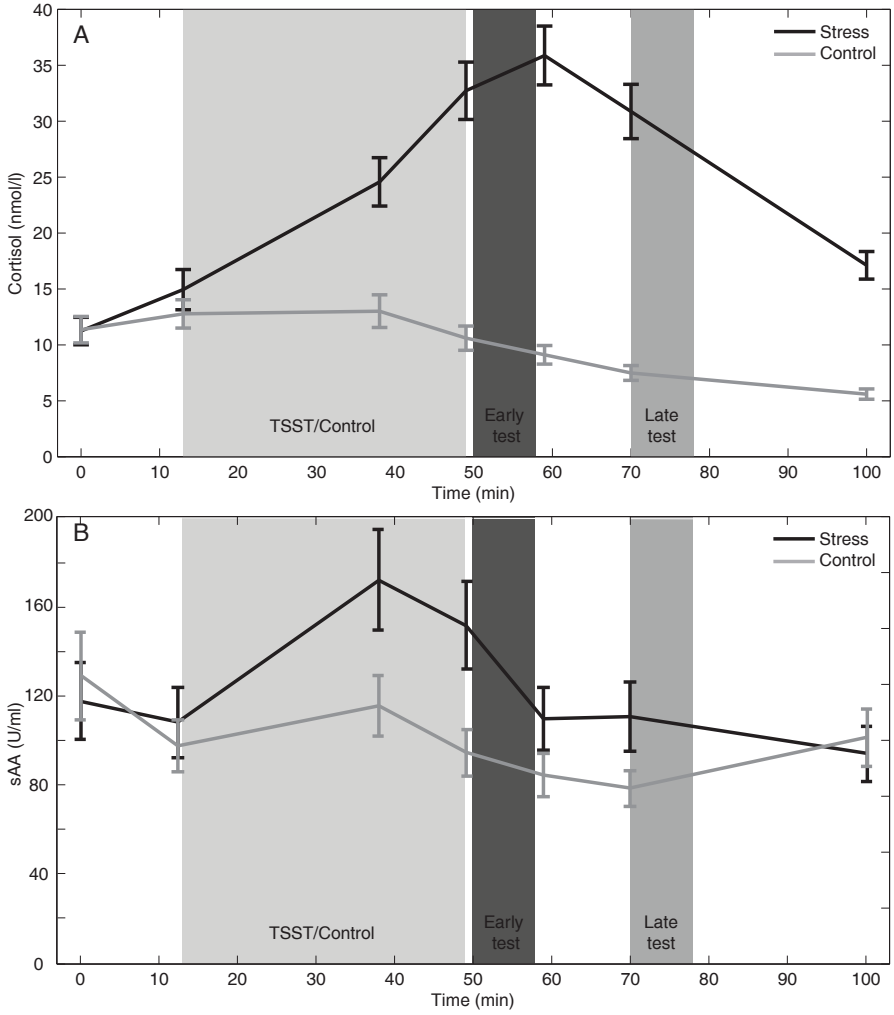

Figure 3.4.1: Timecourse of cortisol activation (A) and salivary alpha amylase (B) throughout the Trier Social Stress Test. The mean of samples 1 and 2 served as baseline samples. Cortisol reactivity to the Trier Social Stress test was defined as area under the curve in the "stress" group with respect to this baseline.

VARIABLES	(1) model1 cortbase	(2) model2 cortbase	(3) model3 cortbase	(4) model4 cortbase
income	-0.00420**	-0.00351**	-0.00324**	-0.00304**
	(0.00174)	(0.00171)	(0.00132)	(0.00143)
timeawake		0.000168	0.000132	0.000148
		(0.000113)	(0.000135)	(0.000155)
age			-0.723	-0.644
			(0.491)	(0.539)
bmi			-0.0350	0.0257
			(0.551)	(0.535)
polright			0.0185	0.00485
			(0.0421)	(0.0509)
numsibs			0.228	-0.104
			(1.138)	(1.458)
recentsmoke				-1.506
				(3.439)
recenteat				2.502
				(2.628)
recentalcohol				-1.080
				(3.259)
Constant	15.36***	19.00***	33.16	30.82
	(1.612)	(3.145)	(21.96)	(22.39)
Observations	79	73	72	70
R-squared	0.065	0.086	0.106	0.132

Table 3.4.1: Baseline cortisol and income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 cortbase	(2) model2 cortbase	(3) model3 cortbase	(4) model4 cortbase
lnincome	-3.280**	-3.060*	-2.859*	-2.912*
	(1.632)	(1.732)	(1.699)	(1.674)
timeawake		0.000147	0.000126	0.000133
		(0.000114)	(0.000129)	(0.000148)
age			-0.568	-0.509
			(0.459)	(0.529)
bmi			-0.193	-0.117
			(0.563)	(0.546)
polright			0.00381	-0.00967
			(0.0442)	(0.0493)
numsibs			0.110	-0.182
			(1.052)	(1.309)
recentsmoke				-0.784
				(3.344)
recenteat				2.769
				(2.514)
recentalcohol				-1.074
				(3.140)
Constant	33.07***	35.18***	49.49*	47.25*
	(10.50)	(10.53)	(25.40)	(25.39)
Observations	79	73	72	70
R-squared	0.099	0.130	0.144	0.175

Table 3.4.2: Baseline Cortisol and Log Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 cort_AUCi	(2) model2 cort_AUCi	(3) model3 cort_AUCi	(4) model4 cort_AUCi
income	0.150	0.233**	0.446***	0.434***
	(0.155)	(0.107)	(0.133)	(0.148)
timeawake		0.0234	0.0423*	0.0479*
		(0.0188)	(0.0232)	(0.0268)
age			37.21	15.99
			(105.8)	(116.0)
bmi			80.73	81.66
			(60.00)	(65.74)
polright			-23.09**	-23.69**
			(9.478)	(11.16)
numsibs			-206.2	-219.8
			(141.0)	(178.4)
recentsmoke				157.8
				(385.5)
receneat				18.47
				(336.9)
o.recentalcohol				0
				(0)
Constant	673.5***	1,383**	98.29	724.7
	(182.4)	(530.4)	(2,389)	(2,700)
Observations	40	34	33	31
R-squared	0.009	0.056	0.285	0.281

Table 3.4.3: Cortisol Stress Reactivity and Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 cort_AUCi	(2) model2 cort_AUCi	(3) model3 cort_AUCi	(4) model4 cort_AUCi
lnincome	98.60	216.1***	302.9***	299.2***
	(84.04)	(78.12)	(87.39)	(102.6)
timeawake		0.0301	0.0469*	0.0524*
		(0.0198)	(0.0235)	(0.0272)
age			3.016	-14.91
			(100.9)	(110.9)
bmi			111.0*	114.5
			(58.00)	(67.22)
polright			-19.80**	-20.90**
			(7.970)	(9.107)
numsibs			-158.6	-168.7
			(142.7)	(184.3)
recentsmoke				82.20
				(303.0)
recenteat				72.75
				(284.1)
o.recentalcohol				0
				(0)
Constant	163.5	401.6	-1,453	-943.4
	(502.8)	(456.3)	(2,174)	(2,531)
Observations	40	34	33	31
R-squared	0.011	0.094	0.322	0.319

Table 3.4.4: Cortisol Stress Reactivity and Log Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

No relationship between income and sAA AUC was found, suggesting that income is not associated with stress reactivity in terms of sAA.

3.5 Discussion

The purpose of this paper was to assess whether low income is associated with high baseline cortisol or sAA levels, or differential stress reactivity of cortisol or sAA as a function of income. We found that low-income subjects indeed show higher baseline levels of cortisol, and blunted responsivity of cortisol to a laboratory stress test, compared to richer participants. In contrast, no effects on income on either baseline sAA levels or sAA responsivity to stress were found. These results expand the existing literature in several ways. First, previous studies have yielded conflicting results regarding the association between baseline cortisol and income: some studies found a negative correlation, as we do here (Cohen et al., 2006a, 2006b; Evans & Kim, 2007; Evans et al., 2000; Li et al., 2007; Lupien et al., 2000; Arnetz et al., 1991), while others find no association or mixed results for different subgroups of participants or aspects of the diurnal cortisol profile (Dowd et al., 2006; Gersten, 2008; Goodman et al., 2005; Ranjit et al., 2005; Rosmond & Bjorntorp, 2000; Steptoe et al., 2005; Decker, 2000; Rosero-Bixby & Dow, 2009), or a positive correlation (Brandstadter et al., 1991; Chen & Paterson, 2006; Fiocco et al., 2007). Our study adds to this literature by showing that baseline levels of cortisol taken in the afternoon in healthy male undergraduate students strongly predict their income. Closer comparison of our study with those which produced conflicting results suggests potential explanations: we find our association between baseline cortisol and income in afternoon levels, and some previous studies reporting null results measured overnight cortisol (Dowd & Goldman, 2006; Gersten, 2008) or morning levels (Goodman et al., 2005). Also, some studies that report null results or associations in the other direction use children or teenage samples, replacing own income with parental education (Goodman et al., 2007) or neighborhood SES (Chen & Paterson, 2006); it may be the case that the association between cortisol and SES only emerges in adulthood when using own income as a predictor. Furthermore, instead of income, some studies use self-reported stress about one's financial situation, occupational status, or a discretized income variable, such as quartiles, all of which are likely to be less fine-grained measures than the continuous income variable we use here (Dowd & Goldman, 2006; Gersten, 2008; Rosmond & Bjorntorp, 2000). Finally, one study reporting a null result has only 30 subjects (Decker, 2000), and two do not report cortisol as a separate outcome, but in combination with other measures such as

VARIABLES	(1) model1 saabase	(2) model2 saabase	(3) model3 saabase	(4) model4 saabase
income	0.00990	0.0102	0.0131	0.0122
	(0.0171)	(0.0174)	(0.0170)	(0.0151)
timeawake		0.000496	3.83e-06	-0.000666
		(0.00155)	(0.00181)	(0.00180)
age			-3.370	-6.481
			(7.779)	(9.179)
bmi			-9.048	-9.509
			(5.992)	(5.977)
polright			0.186	0.233
			(0.511)	(0.615)
numsibs			-1.676	3.821
			(16.58)	(15.71)
recentsmoke				68.68
				(63.04)
recenteat				-35.32
				(22.03)
recentalcohol				-36.40*
				(20.67)
Constant	109.7***	124.8***	384.7	441.7
	(15.07)	(44.23)	(233.9)	(268.2)
Observations	79	73	72	70
R-squared	0.003	0.003	0.037	0.118

Table 3.4.5: Baseline sAA and Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 saabase	(2) model2 saabase	(3) model3 saabase	(4) model4 saabase
lnincome	6.314	6.958	5.983	4.598
	(13.01)	(13.77)	(11.97)	(11.57)
timeawake		0.000498	-0.000141	-0.000823
		(0.00154)	(0.00177)	(0.00174)
age			-3.414	-6.431
			(7.810)	(9.176)
bmi			-8.455	-8.997
			(5.892)	(5.903)
polright			0.239	0.289
			(0.524)	(0.613)
numsibs			-0.803	4.799
			(16.28)	(15.35)
recentsmoke				68.65
				(62.82)
recenteat				-35.97
				(22.17)
recentalcohol				-34.67*
				(19.95)
Constant	76.68	88.09	336.9	401.1
	(80.48)	(86.66)	(235.1)	(276.0)
Observations	79	73	72	70
R-squared	0.003	0.003	0.035	0.116

Table 3.4.6: Baseline sAA and Log Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 sAA_AUCi	(2) model2 sAA_AUCi	(3) model3 sAA_AUCi	(4) model4 sAA_AUCi
income	0.608	0.946	1.036	1.109
	(0.792)	(0.640)	(0.931)	(1.029)
timeawake		0.0900	0.121	0.137
		(0.0983)	(0.113)	(0.147)
age			-239.0	-241.7
			(486.5)	(583.8)
bmi			-376.2	-299.7
			(268.7)	(335.1)
polright			-29.54	-40.22
			(46.06)	(55.95)
numsibs			72.85	234.7
			(1,046)	(1,297)
recentsmoke				-1,491
				(1,753)
receneat				1,456
				(1,537)
o.recentalcohol				0
				(0)
Constant	1,547*	3,779	18,840	17,371
	(836.5)	(2,846)	(14,152)	(19,473)
Observations	40	34	33	31
R-squared	0.007	0.036	0.124	0.147

Table 3.4.7: sAA Stress Reactivity and Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 sAA_AUCi	(2) model2 sAA_AUCi	(3) model3 sAA_AUCi	(4) model4 sAA_AUCi
lnincome	27.32	252.3	60.03	93.34
	(573.8)	(620.1)	(697.3)	(778.7)
timeawake		0.0901	0.0969	0.109
		(0.103)	(0.123)	(0.161)
age			-296.1	-291.4
			(479.6)	(656.6)
bmi			-366.1	-297.3
			(289.9)	(358.8)
polright			-16.13	-22.58
			(45.38)	(57.34)
numsibs			280.0	462.2
			(1,048)	(1,345)
recentsmoke				-933.4
				(1,976)
recenteat				1,346
				(1,686)
recentcaffeine				-390.1
				(2,248)
o.recentalcohol				0
				(0)
Constant	1,735	2,813	18,770	16,928
	(3,465)	(3,703)	(14,801)	(21,063)
Observations	40	34	33	31
R-squared	0.000	0.019	0.104	0.126

Table 3.4.8: sAA Stress Reactivity and Log Income. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

epinephrine, or "at-risk" factors such as BMI (Gersten, 2008; Rosero-Bixby & Dow, 2009). Thus, it is likely that the significant relationship between baseline cortisol and income that we observe here is due to careful isolation of afternoon cortisol levels, together with our fine-grained income measure.

Second, this study is the first to rigorously show blunted cortisol reactivity to stress in low-income subjects. A previous study by Kristenson et al. (2001) found a similar result, but compared subjects from Lithuania (low SES) to subjects from Stockholm (high SES); this cross-country comparison naturally has a host of potential confounds and is thus less rigorous than the within-subject pool regression on monthly income we report here. Intriguingly, Fiocco et al. (2007) found a higher cortisol response to the TSST in subjects with low compared to high education; since education and income are usually positively correlated, this finding appears to contradict that of the present study. Since we used a sample of university students who were almost identical in educational achievement, we could not fruitfully address the respective relationships between income and cortisol reactivity vs. education and cortisol reactivity; further studies with more diverse samples will be required to answer this question.

Third, we find no associations between baseline sAA and income, or sAA reactivity to stress and income. To our knowledge, this is the first study of the relationship between baseline sAA and income in adults. In children, Granger et al. (2006) reported a negative correlation between baseline sAA levels and SES, while Wolf et al. (2008) found a similar relationship in asthmatic but not healthy children. Since these results are conflicting and we are the first to have addressed this question with adults, and with a relatively small sample, we suggest that future studies revisit the relationship between baseline sAA and income.

Finally, this is the first report on the relationship between income and sAA stress reactivity; we find no significant association. However, in our view the hypothesis that sAA stress reactivity may be related to income remains plausible, as a number of previous studies have found blunted stress reactivity in terms of other sympathetic markers, in particular cardiovascular variables such as heart rate and blood pressure (Owens et al., 1993; Lynch et al., 1998; Carroll et al., 1997, 2000; Steptoe et al., 2002; Brydon et al., 2004). Thus, future studies might revisit the question of whether low-SES subjects may have altered sAA stress reactivity.

What is the physiological and behavioral significance of higher baseline cortisol levels, and blunted cortisol reactivity to stress, in low-income subjects? First, higher baseline cortisol levels in low-SES subjects may contribute to the higher rates or morbidity and mortality in this population. Specifically, chronically elevated cortisol levels have been shown to lead to

hippocampal atrophy and memory deficits (Lupien et al., 1998), increased risk of cardiovascular disease and stroke (Rosmond & Bjorntorp, 2000), and immunosuppression (Cacioppo et al., 2002). Elevated baseline cortisol levels are also observed in chronically stressed rats (Katz, 1981) and monkeys (Sapolsky, 1993), as well as human patients suffering from depression (Holsboer, 2000; Checkley, 1996). Second, altered cortisol reactivity has been associated with a host of debilitating conditions, such as chronic stress (Kristenson et al., 1998; Benschop et al., 1994; van der Pompe, 1996), effort-reward imbalance (Siegrist et al., 1997), and vital exhaustion (Nicholson & van Diest, 2002); smokers and alcoholics also show attenuated cortisol reactivity to stress (Errico et al., 1993; Kirschbaum et al., 1993; Roy et al., 1994). In sum, the cortisol baseline and reactivity differences that we here associate with low income are also associated with a host of adverse psychosocial and health outcomes. Of course, at present it remains unclear in which direction causality runs: the cortisol differences we observe could be a cause or a consequence of low income, or even of any of the psychosocial and socioeconomic factors mentioned above which are usually associated with poverty. What remains, however, is that once the cortisol differences have been established, they lead to adverse health consequences, and thus are one possible channel that may account for the link between low income and morbidity and mortality (Steptoe & Marmot, 2002; Baum et al., 1999; Brunner, 1997; Kristenson et al., 2004).

A less well established, but potentially even more intriguing channel through which altered cortisol baselines and reactivity may affect long-term outcomes is through immediate behavioral consequences. It has long been known that chronically elevated cortisol levels have adverse consequences for memory processes (for reviews, see McEwen & Seeman, 1999; McEwen, 2004; McEwen & Sapolsky, 1995; de Kloet et al., 1999; Lupien et al., 2007, 2009; Kim & Haller, 2007). In line with these findings and the present results, Evans & Schamberg (2009) found that working memory performance in young adults was lower for individuals coming from poor families; moreover, the coefficient on poverty became non-significant when the authors controlled for allostatic load during childhood. Allostatic load was a composite measure which included overnight urine levels of cortisol, epinephrine, and norepinephrine, body mass index, and resting blood pressure. This finding suggests that altered cortisol levels may directly contribute to the adverse cognitive long-term outcomes that are typically observed in children from poor backgrounds.

Furthermore, a number of studies now show that economic decision-making is impaired by increased cortisol levels. Specifically, subjects with (experimentally) raised cortisol levels subjects perform worse on verbal anal-

ogy tasks (Keinan, 1987) and are less able to learn the optimal response among several available options (Gray, 1999; Preston et al., 2007; van den Bos et al., 2009). Recently, Porcelli and Delgado (2009) found that stressed subjects are less rational in risky decision-making, showing a greater degree of risk aversion in the gain domain, and of risk seeking in the loss domain, than non-stressed subjects. In our own work, we recently found that subjects with strongly increased cortisol levels became more present-biased in making choices between amounts of money available at different times (Cornelisse et al., in preparation).

In sum, we have shown that poor individuals have higher baseline cortisol levels, and blunted cortisol reactivity to stress, than richer individuals. Salivary alpha amylase showed no relationship to income, neither in terms of baseline levels nor stress reactivity. Together, these findings substantiate the claim that poverty is characterized by particular neurobiological consequences; these consequences, in turn, may have adverse consequences for health and decision-making, and thus contribute to exacerbating the poverty that precipitated them.

Bibliography

[1] Arnetz, B.B., Brenner, S.O., Levi, L., Hjelm, R., Petterson, I.L., Wasserman, J., Petrini, B., Eneroth, P., Kallner, A., Kvetnansky, R., et al. (1991). Neuroendocrine and immunologic effects of unemployment and job insecurity. Psychotherapy and Psychosomatics, 55, 76-80.

[2] Baum A, Garofalo JP, Yali AM. (1991). Socioeconomic status and chronic stress: does stress account for SES effects on health? Ann NY Acad Sci, 896, 131–44.

[3] Benschop, R., Brossehot. J., & Ballieux, R. (1994). Chronic stress affects immunologic but not card tova.˜ eular responsiveness to acute stress in humans. American Jvnrnal of Physiology. 266, R75-R90.

[4] Brunner E. Stress and the biology of inequality. Br Med J 1997;314:1472–76.

[5] Brydon, L., Edwards, S., Mohamed-Ali, V., Steptoe, A., 2004. Socioeconomic status and stress-induced increases in interleukin-6. Brain, Behavior and Immunity 18, 281–290.

[6] Cacioppo JT, Kiecolt-Glaser JK, Malarkey WB et al. Autonomic and glucocorticoid associations with the steady-state expression of latent Epstein-Barr virus. Hormones Behav 2002;42:32–41.

[7] Carroll D, Davey Smith G, Sheffield D, Shipley MJ, Marmot MG (1997). The relationship between socioeconomic status, hostility, and blood pressure reactions to mental stress in men: data from the Whitehall II study. Health Psychol, 16, 131–6.

[8] Carroll D, Harrison LK, Johnston DW et al. Cardiovascular reactions to psychological stress: the influence of demographic variables. J Epidemiol Comm Health 2000; 54: 876–7.

[9] Checkley, S. (1996). The neuroendocrinology of depression and chronic stress. British Medical Bulletin, 52, 597–617.

[10] Cohen S., Doyle, W.J., & Baum, A. (2006a). Socioeconomic status is associated with stress hormones. Psychosom Med, 68, 414-420.

[11] Cohen, S., Schwartz, J., et al. (2006b). Socioeconomic status, race and diurnal cortisol levels in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Psychosom Med, 68, 41-50.

[12] Dowd, J.B., Simanek, A.M., & Aiello, A.E. (2009). Socio-economic status, cortisol and allostatic load: a review of the literature. International Journal of Epidemiology, 38, 1297–1309.

[13] Ehlert, U., Erni, K., Hebisch, G., Nater, U., 2006. Salivary alpha-amylase levels after yohimbine challenge in healthy men. The Journal of Clinical Endocrinology and Metabolism 91, 5130–5133.

[14] Errico, A., Parsons, O, King, A., & Lovallo, W. (1993). Attenuated eortisoI response to biobehavioural stressors in sol:mr alcoholics. Jo*ernal if Studies on Alcohol, 54, 393-39g,

[15] Evans, G.W., & Kim, P. (2009). Childhood poverty and health: cumulative risk exposure and stress dysregulation. Psychological Science, 2007, 18, 953–57.

[16] Evans, G.W., & English, K. (2002). The environment of poverty: multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. Child Development, 2002, 73, 1238–1248.

[17] Holsboer, F. (2000). The Corticosteroid Receptor Hypothesis of Depression. NEUROPSYCHOPHARMACOLOGY 23, 477-501.

[18] Katz, R, J, (1981) Acute and chronic stress effects on open field activity in the rat: lrnplieafions for a model of depression. Neurt˜scrence Brobehm'ioral Revtews, 5, 2,˜7-251.

[19] Kirschbaum, C., Hellhammer, D.H., 1994. Salivary cortisol in psychoneuroendocrine research: recent developments and applications. Psychoneuroendocrinology 19, 313–333.

[20] Kirschbaum, C., Pirke, K.M., Helhammer, D.H. Neuropsychobiology 28, 76-81 (1993).

[21] Kirschbaurn, C., Strasburger, J., & Langkrär, J. (1993). Attenuated cortisol response to psychological stress but not to CRH or ergometry in young habitual smokers. Pharmacol Biochem Behav, 44, 527-531.

[22] Kristenson M, Kucinskienë Z, Bergdahl B, Calkauskas H, Urmonas V, Orth-Gomér K. (1998). Increased psychosocial strain in Lithuanian versus Swedish men: the LiVicordia study. Psychosom Med., 60, 277-82.

[23] Kristenson M, Eriksen HR, Sluiter JK, Starke D, Ursin H. Psychobiological mechanisms of socioeconomic differences in health. Soc Sci Med 2004;58:1511–22.

[24] Li, L., Power, C., Kelly, S., Kirschbaum, C., & Hertzman, C. Life-time socio-economic position and cortisol patterns in mid-life. Psychoneuroendocrinology, 2007, 32, 824–33.

[25] Lupien SJ, de Leon M, de Santi S et al. Cortisol levels during human aging predict hippocampal atrophy and memory deficits. Nat Neurosci 1998;1:69–73.

[26] Lynch JW, Everson SA, Kaplan GA et al. Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis. Am J Public Health 1998; 88: 389–94.

[27] Nicolson, N. A., & van Diest, R. (2000). Salivary cortisol patterns in vital exhaustion. Journal of Psychosomatic Research, 49, 335–342.

[28] Owens JF, Stoney CM, Matthews KA. Menopausal status influences ambulatory blood pressure levels and blood pressure

[29] Patton, J.H., Stanford, M.S. and Barratt, E.S. J Clin Psychol 51, 768-774 (1995).

[30] Rosmond R, Bjorntorp P. The hypothalamic–pituitary–adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes and stroke. J Int Med, 247, 188–97.

[31] Roy, M.P., Steptoe, A., & Kirschbaum, C. (1994). Association between smoking status and cortisol stress responsivity in healthy young men. International Journal of Behavioral Medicine, 1, 264-283.

[32] Sapolsky. R (1993). Endocrinology Alfresco. Psychoendocrine studies of wild baboons, Recent Progress in Hormone Research, 48, 437-468.

[33] Siegrist, J., Klein, D., & Voigt, K.-H. (1997). Linking sociological with physiological data: The model of effortreward imbalance at work. Acta Physiologica Scandinavica, 161, 112–116.

[34] Steptoe A, Marmot M. The role of psychobiological pathways in socio-economic inequalities in cardiovascular disease risk. Eur Heart J 2002;23:13–25.

[35] Steptoe, A., Feldman, P.J., Kunz, S., Owen, N., Willemsen, G.H., Marmot, M., 2002. Stress responsivity and socioeconomic status. A mechanism for increased cardiovascular risk? Euro. Heart J. 23, 1757–1763.

[36] van der Pompe, G., Antoni, M. H., & Heijnen, C. J (1996). Elevated basal eortisol levels and attenuated ACTH and cortisol responses to a behavioral challenge in women with metastatic breast cancer. Psychoneuroendocrinology, 21, 361-374,

[37] van Stegeren, A., Rohleder, N., Everaerd, W., Wolf, O.T., 2006. Salivary alpha amylase as marker for adrenergic activity during stress: effect of betablockade. Psychoneuroendocrinology 31, 137–141.

[38] Von Dawans, B., Kirschbaum, C. & Heinrichs, M. (2011). The Trier Social Stress Test for Groups (TSST-G): A new research tool for controlled simultaneous social stress exposure in a group format. Psychoneuroendocrinology, 36, 514-522.

Chapter 4

Poverty Raises Levels of the Stress Hormone Cortisol: Evidence from Weather Shocks in Kenya

4.1 Summary

Does poverty lead to stress? Despite numerous studies showing correlations between socioeconomc status and levels of the stress hormone cortisol, it remains unknown whether this relationship is causal. In two studies, we used random weather shocks in Kenya to address this question. First, we study the effects of a severe drought in Kajiado district in 2008/2009; during this drought, the exclusively Maasai population of the region lost 41.5% of their goats and sheep on average. We show evidence that livestock deaths were not predicted by observable characteristics, and therefore represent a random shock to the households. A year later, in 2010, we observe a strong relationship between the proportion of goats and sheep that a household lost to the drought, and both the husbands' and wives' baseline levels of the stress hormone cortisol. In the second study, we obtained salivary cortisol samples from poor rural farmers in Kianyaga district, Kenya, together with GPS coordinates for household location, and high-resolution infrared satellite imagery meausring rainfall. Since rainfall is the main input into agricultural productivity in the region, the absence of rain constitutes a random negative income shock. We find that low levels of rain strongly raise cortisol levels with a temporal lag of 10-20 days. Together, these findings suggest that increases in poverty lead to increases in the stress hormone cortisol.

4.2 Introduction

More than 1 billion people in the world still live below the "extreme poverty" line of $25 a day defined by the World Bank (2007). The economic and health consequences of material poverty are well-known; it brings with it malnutrition, lack of shelter, insufficient health care, low life expectancy, and poor access to education. However, the psychological consequences have received less attention; nevertheless, in recent years, a small literature has emerged that asks whether poverty also has psychological and neurobiological consequences.

A prominent hypothesis in this domain is that poverty may lead to increases in stress, and in particular the stress hormone cortisol. This hypothesis is supported by several lines of evidence. First, recent work in the psychology and economics of happiness has documented a robust relationship between income and happiness, both within and across countries: poor people are less happy and satisfied with their lives than rich people in the same country; in addition, people in richer countries are, on average, happier than people in poorer countries (Stevenson & Wolfers, 2008; see also Easterlin, 1974).

Conversely, the prevalence of depression in developing countries is staggering: while the prevalence rates in Europe and North America hover between 5-10%, developing countries report numbers such as 19% (Lebanon, Mexico), 20% (Thailand), 24% (Uganda), 39% (Dominican Republic) and 40% (Cuba; Bolton et al., 2004; Garcia-Alvarez, 1986; Thavichachart et al., 2001; Sobocki et al., 2006; Patel et al., 2003). This finding supports the putative relationship between poverty and stress because stress is a significant factor in the etiology of depression: 80% of all patients with depression have histories of chronic stress or stressful life events (Hammen, 2005), and depression is marked by dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, which controls the release of cortisol (Holsboer, 2000).

Finally, a number of authors have argued that low-income environments may be characterized by both greater exposure to stressful events, and the absence of resources to deal with such stress (Baum et al., 1999; Steptoe et al., 2002; Brunner, 1997; Kristenson, 2004).

Together, these strands of literature suggest that poverty may be characterized by increased levels of stress, and in particular the stress hormone cortisol. Indeed, this relationship has been confirmed in a number of studies which find significant correlations between socio-economic status (SES), self-reported stress, and cortisol (Cohen et al., 2006a, 2006b; Evans & Kim, 2009; Evans & English, 2002; Li et al., 2007; Lupien et al., 2000; Arnetz et al., 1991; see Dowd et al., 2009, for a review). Note, however, these studies

were conducted in developed countries; it remains unclear whether a similar relationship exists in developing countries. More importantly, these findings are merely correlational and therefore do not justify conclusions about whether poverty causes stress or vice-versa.

The present study aims to fill this gap. Using weather shocks in two regions of Kenya, we identify a causal effect of increases in poverty on increases in the stress hormone cortisol. Specifically, we show that Maasai tribespeople in Kajiado district, who were exposed to a severe drought in 2008/2009 which caused the death of a large number of livestock, show higher cortisol levels in 2010 depending on how many goats and sheep they lost during the drought. Second, we use high-resolution satellite rainfall data together with household-level GPS and cortisol data from Kikuyu farmers in Kinayaga district to show that farmers have higher levels of cortisol if the previous 10-20 days brought low amounts of rain. Together, our findings establish a causal relationship between exogenous increases in poverty and levels of cortisol.

4.3 Materials and Methods

4.3.1 Subjects and Setting

We studied 152 Maasai tribespeople (77 women) in Kajiado distict, Kenya, and 283 Kikuyu farmers (115 women) in Kianyaga district, Kenya, between January and December 2010. Each participant gave written consent; illiterate participants gave consent by fingerprint. The study was approved by the ethics commissions at the University of Zurich, McGill University, Innovations for Poverty Action Kenya (IPAK), and the Kenya Medical Research Institute (KEMRI). Participants received KSH 100 (USD 1) for participation; in addition, they could earn money in the economic games that were part of the questionnaire.

4.3.2 Procedure

In Kajiado district, data were collected in three locations: Elangata Wuas, Torosei, and Kilonito. Households were chosen by random sampling based on a list of households obtained from a previous round of surveys in 2008. New households were added to the sampling frame and also chosen at random. In Kianyaga, households were chosen randomly based on household lists obtained from village elders.

In both locations, data were collected in one-on-one field interviews at the respondents' homestead by trained enumerators. Interviews were conducted

in Maa, the tribal language of the Maasai, in Kajiado district, and in Kikuyu in Kianyaga district. To ensure accurate translations of the questionnaire, it was translated into Maa and Kikuyu by four different translators, and then back-translated into English by another four translators. The four back-translated versions were then compared to the English original, and the team of 8 translators plus one supervisor agreed on a final translation.

At the beginning of the interview, the consent script was read and consent was obtained by signature or as a thumbprint. Respondents were paid after completing the interview. Saliva samples were obtained before and after the interview.

4.3.3 Questionnaire and GPS data

In both locations, we administered a standard socioeconomic questionnaire that elicited information about household structure, income, education, health, and current worries. In Kajiado district, the questionnaire also asked about the loss of livestock during the 2008/2009 drought; specifically, respondents were asked how many cows, goats and sheep they owned before the drought, how many died during the drought for reasons related to the drought, how many died or left the herd for other reasons, and how many animals they owned currently. Since livestock is the main source of wealth among the Maasai, respondents knew very well how many animals they owned and how many were lost during the drought.

The questionnaire data from Kianyaga is still being cleaned, and thus only a subset of the variables are available for analysis. However, for the purpose of this paper, the crucial data is the GPS coordinates of the households, which are available. They were collected using a handheld GPS device and recorded in degrees of latitude and longitude, at a resolution of 1/1000th of an arcminute, which corresponds to ~0.18 meters at this proximity to the equator.

4.3.4 Rainfall data

Rainfall data were obtained from the Famine Early Warning Systems Network, FEWSNET (www.fews.net). The data were originally downloaded in ArcGIS format, and then transferred into Stata format using a custom-written FORTRAN program. The data are provide a rainfall estimate based on high-resolution Meteosat infrared data, rain gauge reports from the global telecommunications system, and microwave satellite observations. The data cover the years 2000-2011; for the purpose of this study, the relevant time frame is from 20 days before our surveys began until they ended (see below

for choice of lag for the rain timeseries), i.e. December 2009 – December 2010. The data are dekadal, i.e. averaged over 10-day intervals. The spatial resolution is 0.1°, which corresponds to 11 km at this proximity to the equator. To obtain household-specific rainfall data, the satellite rainfall data was first subjected to bilinear interpolation, and then combined with the GPS location of each household. This yielded a rainfall estimate that was unique to each household, for each 10-day interval in the study period.

4.3.5 Salivary cortisol

Salivary samples were obtained using Salivette sampling devices (Sarstedt, Nümbrecht, Germany), once before and once after questionnaire administration. Salivary samples were stored at room temperature for at most 10 days, and then transported to Nairobi, where they were stored at -20°C until further analysis. Free cortisol levels were measured at Lancet Pathologists, Nairobi. In a blinded test of this laboratory with duplicate samples, the correlation across sample pairs was r = 0.995 (N=60). Free cortisol is the physiologically active component of cortisol, and is closely related to the rate of cortsiol secretion by the adrenal gland (Kirschbaum & Hellhammer, 1989; Aardal & Holm, 1995; Aardal-Ericsson et al., 1998). During analysis, the two samples were average to obtain more stable estimates of cortisol levels. The cortisol data were pre-processed by regressing out the logarithm of time since waking and keeping only the residuals from this regression; the resulting cortisol data can thus be interpreted as the deviations from the typical declining diurnal cortisol profile (van Cauter et al., 1996).

4.3.6 Statistical analysis

4.3.6.1 Autoregressive Order Selection and Timeseries Order of Integration

The rainfall data is likely to contain serial correlation. We model this serial correlation as an autoregressive process. We use information criteria to choose the most appropriate autoregressive order for the rainfall process. We report both the Akaike Information Criterion (Akaike, 1973, 1974, 1978) and Schwarz's Bayesian information criterion (SBIC; Schwarz, 1978).

To ensure stationarity of the time series, we test for the presence of a unit root using the augmented Dickey-Fuller Test (Elliott et al., 1996). The absence of a unit root implies stationarity of the time series and integration of order 0. The null hypothesis of the Dickey-Fuller test is the presence of a unit root; a large negative value rejects this hypothesis. We perform the test

using the autoregressive order determined with information criteria, both with and without lag and drift terms.

4.3.6.2 Regression Specifications

To establish whether the proportion of goats and sheep that died as a result of the drought can be considered an exogenous shock, we first regress this variable on a number of observables using OLS. Specifically, we use the following model on the data from Kajiado district:

$$prop\_goatssheep\_died\_total_i = \beta_0 + \beta_1 familyincome30_i$$
$$+\beta_2 goatssheet\_before_i + \beta_3 female_i + \beta_4 numkidsown_i$$
$$+\beta_5 married_i + \beta_6 schooling_i + u_i,$$

where $prop\_goatssheep\_died\_total_i$ indicates what proportion of goats and sheep owned by the respondent died during the 2008/2009 drought, $familyincome30_i$ is family income over the last 30 days in Kenyan Shillings (KES; 1 KES $\approx$ 1 USD, October 2011), $goatssheep\_before_i$ is the number of goats and sheep owned by the respondent before the 2008/2009 drought, $female_i$ is a dummy variable for gender, $numkidsown_i$ is the number of own children living in the household, $married_i$ is a dummy variable for marital status, $schooling_i$ is education in years of schooling, and u_i is the error term.

Second, to test whether cortisol was affected by the proportion of goats and sheep that died as a result of the drought, we run the following OLS regression on the data from Kajiado district:

$$cort_i = \beta_0 + \beta_1 prop\_goatssheep\_died\_total_i + \beta_2 goatssheep\_before_i$$
$$+\beta_3 goatssheep\_after_i + \beta_4 female_i + \beta_5 sick7_i + \beta_6 numkidsown_i +$$
$$+\beta_7 married_i + \beta_8 schooling_i + \beta_9 familyincome30_i + u_i,$$

where $cort_i$ is the respondent's salivary cortisol level, $sick_7$ indicates whether the respondent was ill in the previous week, and the remaining variables are as described above. Our main research question is whether the coefficient on $prop\_goatssheep\_died\_total_i$ is significantly positive; this would indicate that larger losses of livestock lead to higher cortisol levels a year later.

We run both of these regressions using heteroskedasticity-robust standard errors; in addition, we correct the standard errors for clustering by Enkang, which is the small group of houses in which Maasai live.

Finally, to assess the effect of rainfall shocks on cortisol levels, we run the following regression on the data from Kianyaga district:

$$cort_{i,t} = \beta_0 + \beta_1 rain_{i,t} + \beta_2 rain_{i,t-1} + \beta_3 female + u_i,$$

where t is the dekad subscript, $rain_{i,t}$ is the household-specific rainfall measure at time t, and the remainder of the variables are as described above. Modeling rainfall as an AR(1) process reflects the results from the autoregressive order selection procedure, reported below. Again we use heteroskedasticity-robust standard errors. The coefficients of interest are those on the current and lagged rainfall measures: if negative weather shocks contribute to stress and raise cortisol levels, we should observe negative coefficients on these variables.

To assess the overall effect of rain on cortisol in this model, we perform an F-test for joint significance of the current and lagged rainfall measures.

4.4 Results

4.4.1 Kajiado: Livestock loss and cortisol

Our first question was whether the loss of livestock due to the 2008/2009 drought in Kajiado district had led to an increase in cortisol levels. Figure 4.4.1 shows the monthly average rainfall, in mm, in Kajiado district between January 2008 and December 2009. The grey line indicates the long-run average rainfall per month, in mm, in Kajiado district, between 2003-2007; the black line indicates monthly rainfall in the drought year 2008/2009. The peaks around November/December and April/May represent the short and long rains, respectively. It can be seen that the short rains in 2008 and the long rains in 2009 were significantly reduced in magnitude with respect to the long-run average. Thus, repsondents in this region faced a severe drought during this period.

Table 4.4.1 shows statistics that summarize the effect that this drought had on the livelihoods of our 152 respondents in Kajiado district. Most strikingly, households lost an average of 41.5% of their goats and sheep due to the drought. This is a serious negative shock for these families, as livestock is the main source of income and wealth for the Maasai. Moreover, this is not counting sheep which left the herd for different reasons such as being slaughtered, given away, or lost. This fact explains why the number of goats and sheep owned by a household after the drought is only 65.17, while it was 201.1 before the drought; this corresponds to a decrease of 67.6% in the number of goats and sheep, suggesting that the drought decimated herds

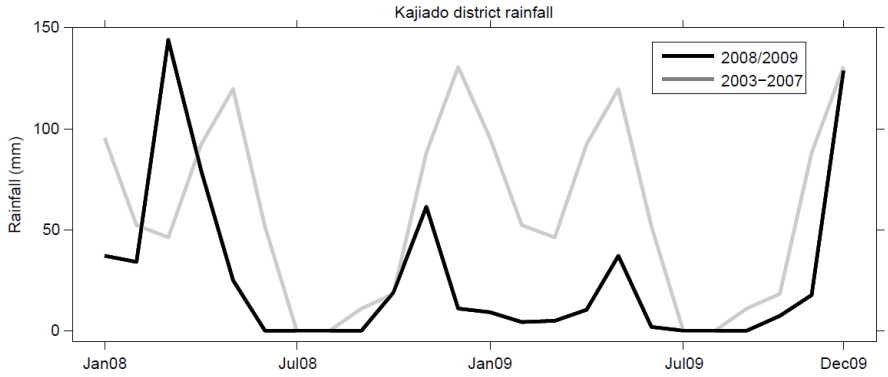

Figure 4.4.1: Rainfall in Kajiado district. The grey line indicates the long-run average rainfall per month, in mm, in Kajiado district, between 2003-2007. The black line indicates monthly rainfall in the drought year 2008/2009. The peaks around November/December and April/May represent the short and long rains, respectively.

through channels other than livestock deaths. Thus, the drought represented an extremely large shock to the economic situation of households in the region.

Importantly, in addition to a large average decrease in the number of goats and sheep due to the drought, we also observe substantial variation: the standard deviation for the proportion of livestock deaths is 0.253, suggesting that not every household was affected equally. This fact creates the possibility of relating the variation in the proportion of livestock lost to later outcomes such as cortisol levels.

To make this relationship causal, we need ot establish first whether livestock deaths due to the drought can be considered a random negative shock. We therefore regressed the proportion of livestock that a household lost during the drought on a number of observables, including family income in the past 30 days, the number of goats and sheep owned before the drought, a dummy variable for gender and marital status, the number of children of the household, and education level in years of schooling. To be able to consider the proportion of livestock lost to the drought a random shock, we need it to be unrelated to these observables. The results are shown in Tables 4.4.2 and 4.4.3; each column shows an OLS regression of the proportion of goats and sheep lost during the drought on the variables described above. In contrast to Table 4.4.2, Table 4.4.3 includes cluster correction of the standard errors at the Enkang level (the Enkang is the group of houses within one homestead, often belonging to the same family or relatives). It can be seen

VARIABLES	(1) mean	(2) sd	(3) median	(4) N
prop_goatssheep_died_total	0.415	0.253	0.377	152
goatssheep_before	201.1	261.4	117	152
goatssheep_after	65.17	81.31	37.50	152
familyincome30	3,400	7,521	0	152
female	0.507	0.502	1	152
numkidsown	7.044	7.699	6	152
married	0.776	0.418	1	152
schooling	2.678	4.167	0	152

Table 4.4.1: Descriptive statistics, Kajiado district. The first row indicates the proportion of goats and sheep that families lost to the 2008/2009 drought. The second and third rows represent the number of goats and sheep that families owned before and after the drought, respectively. Note that the difference between these numbers does not correspond to the proportion of goats and sheep lost to the drought in the first row, as that figure takes into account only animals lost to the drought, while the number of animals owned after the drought also reflects animals that left the herd for different reasons (slaughtered, given away, lost), and animals that joined the herd (born, given to, purcahsed). The fourth row shows family income over the last 30 days, in Kenyan Shillings (KES). 1 KES $\approx$ 1 USD (October 2011). The next rows show the proportion of women in the sample, the number of own children living in the household, the proportion of married respondents, and average education in years of schooling.

that the proportion of goats and sheep that died during the drought is not predicted by any of the right-hand side variables. Almost all coefficients are non-significant, suggesting that the loss of livestock during the drought was an exogenous shock which affected families randomly; in particular, even rich families could not protect themselves from the loss of goats and sheep during the drought.

Having established that the death of goats and sheep during the drought affected rich and poor households relatively uniformly and thus appears to be a random exogenous shock, we next asked whether cortisol levels a year later (in 2010) were predicted by the proportion of goats and sheep each household lost during the drought. We therefore regressed cortisol levels (averaged across the two samples that were collected from each respondent, see Methods) on the proportion of goats and sheep that died during the drought, and a number of control variables. The results are shown in Tables 4.4.4 and 4.4.5, where again Table 4.4.5 reports the results with Enkang clustering. It can be seen that for all specfications, a strong positive relationship exists between the proportion of goats and sheep that died during the drought, and cortisol levels a year later. In terms of magnitude, the specification which includes the full set of control variables suggests that a 50 percentage point increase in the number of goats and sheep lost to drought leads to 31% increase in cortisol levels (a 2.8 nmol/l increase in cortisol from a baseline of 9.0 nmol/l).

Together, these results suggest that the death of livestock due to drought has a strong effect on cortisol levels a year later. A potential remaining concern with these findings, however unlikely, is that cortisol levels may be endogenous to the proportion of goats and sheep that died during the drought. For instance, one might imagine that unobserved heterogeneity in the sample for mental health problems might affect both cortisol and the proportion of livestock lost during the drought. The next study therefore uses an identification strategy that can even more confidently be said to be completely random with regard to household characteristics: rainfall shocks.

4.4.2 Kianyaga: Rainfall shocks and cortisol

Our second question was whether lack of rainfall in a region of small-scale farmers would increase levels of cortisol. We study this question in the Kianyaga district of Kenya, on the slopes of Mt. Kenya, a region populated by Kikuyu people whose household income and consumption depends heavily on agriculture, and thus, rain. Small fluctuations in rainfall can have potentially serious adverse consequences for the harvest and hence household welfare; we therefore hypothesized that fluctuations in rainfall would lead to

	(1)	(2)	(3)
	model1	model2	model3
VARIABLES	prop_goatssheep_died_total	prop_goatssheep_died_total	prop_goatssheep_died_total
familyincome30	2.09e-06	7.69e-07	1.75e-06
	(2.56e-06)	(2.45e-06)	(2.98e-06)
goatssheep_before		9.25e-05*	1.17e-05
		(5.48e-05)	(8.80e-05)
female			-0.0865**
			(0.0421)
numkidsown			0.00661
			(0.00450)
married			0.0107
			(0.0685)
schooling			-0.00627
			(0.00573)
Constant	0.408***	0.393***	0.412***
	(0.0225)	(0.0252)	(0.0476)
Observations	152	152	152
R-squared	0.004	0.011	0.071

Table 4.4.2: The proportion of goats and sheep lost to the drought does not depend on observables in Kajiado district. Each column shows an OLS regression of the proportion of goats and sheep lost during the drought on a group of observable variables, in particular those relating to family wealth. Almost all coefficients are non-significant, suggesting that the loss of livestock during the drought was an exogenous shock which affected families randomly; in particular, even rich families could not protect themselves from the loss of goats and sheep during the drought. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

	(1)	(2)	(3)
	model1	model2	model3
VARIABLES	prop_goatssheep_died_total	prop_goatssheep_died_total	prop_goatssheep_died_total
familyincome30	2.09e-06	7.69e-07	1.75e-06
	(4.30e-06)	(3.81e-06)	(4.48e-06)
goatssheep_before		9.25e-05	1.17e-05
		(7.73e-05)	(0.000112)
female			-0.0865**
			(0.0342)
numkidsown			0.00661
			(0.00529)
married			0.0107
			(0.0804)
schooling			-0.00627
			(0.00759)
Constant	0.408***	0.393***	0.412***
	(0.0289)	(0.0339)	(0.0493)
Observations	152	152	152
R-squared	0.004	0.011	0.071
Enkang clustering	YES	YES	YES

Table 4.4.3: As Table 4.4.2, but with standard errors clustered by Enkang. Robust clustered standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

VARIABLES	(1) model1 cort	(2) model2 cort	(3) model3 cort	(4) model4 cort
prop_goatssheep_died_total	5.092**	4.886**	5.786**	5.626***
	(2.043)	(2.266)	(2.292)	(2.032)
goatssheep_before		0.00368	0.00339	0.00195
		(0.00342)	(0.00330)	(0.00308)
goatssheep_after		0.00495	0.00626	0.00765
		(0.0116)	(0.0114)	(0.00984)
female			0.757	0.523
			(0.949)	(1.029)
sick7			-0.496*	-0.537*
			(0.288)	(0.314)
numkidsown				0.116
				(0.0945)
married				-0.0583
				(1.529)
schooling				0.146
				(0.121)
familyincome30				-2.82e-05
				(6.50e-05)
Constant	6.933***	5.955***	5.616***	4.973***
	(0.870)	(1.070)	(1.104)	(1.216)
Observations	152	152	152	152
R-squared	0.044	0.090	0.106	0.134

Table 4.4.4: Livestock loss due to drought increases cortisol levels in Kajiado district. Each column shows an OLS regression of baseline cortisol levels, in nmol/l, on the proportion of goats and sheep that households lost during the drought, and other observables. All coefficients on the proportion of goats and sheep lost are positive and highly significant, suggesting that loss of livestock increased cortisol levels a year later. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

VARIABLES	(1) model1 cort	(2) model2 cort	(3) model3 cort	(4) model4 cort
prop_goatssheep_died_total	5.092**	4.886**	5.786**	5.626***
	(2.024)	(2.270)	(2.255)	(1.996)
goatssheep_before		0.00368	0.00339	0.00195
		(0.00368)	(0.00343)	(0.00292)
goatssheep_after		0.00495	0.00626	0.00765
		(0.0122)	(0.0118)	(0.00970)
female			0.757	0.523
			(0.800)	(0.910)
sick7			-0.496*	-0.537*
			(0.277)	(0.320)
numkidsown				0.116
				(0.0950)
married				-0.0583
				(1.432)
schooling				0.146
				(0.113)
familyincome30				-2.82e-05
				(5.17e-05)
Constant	6.933***	5.955***	5.616***	4.973***
	(0.890)	(1.076)	(1.059)	(1.192)
Observations	152	152	152	152
R-squared	0.044	0.090	0.106	0.134
Enkang clustering	YES	YES	YES	YES

Table 4.4.5: As Table 4, but with standard errors clustered by Enkang. Robust clustered standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

lag	AIC	SBIC
0	8.5674324	8.6029573
1	8.4657803*	8.53683*
2	8.4790803	8.5856549
3	8.5134761	8.6555756
4	8.5181849	8.6958093
5	8.5284725	8.7416217
6	8.55317	8.8018441
7	8.5871938	8.8713929
8	8.6155775	8.9353014
9	8.6496751	9.0049239
10	8.6456361	9.0364098
11	8.6712603	9.0975589
12	8.7057429	9.1675663
13	8.7280029	9.2253512
14	8.7592022	9.2920754
15	8.7879705	9.3563686
16	8.8095766	9.4134996
17	8.8424953	9.4819432
18	8.8567348	9.5317075
19	8.811245	9.5217426
20	8.8424854	9.5885079

Table 4.4.6: Lag order selection statistics for rainfall in Kianyaga district. Column 1 reports Akaike Information Criterion, column 2 reports Schwarz's Bayesian Information Criterion (SBIC) for the dekadal rainfall variable. Both information criteria are minimzed at lag 1.

stress, reflected in increased levels of cortisol.

We therefore regressed cortisol levels on lagged values of rainfall data obtained from FEWSNET satellite imagery. To this end, we first analyzed the autoregressive order of the rainfall timeseries using information criteria. Specifically, we computed the Akaike Information Criterion and Schwarz's Bayesian Information Criterion for the timeseries of dekadal rainfall indices, up to a maximum lag of 20 dekads (200 days). Table 4.4.6 reports the AIC and SBIC values resulting from this analysis. Both information criteria are minimized at lag 1. In the following we therefore regress cortisol on rainfall for the current and one previous dekad.

Second, to ascertain that the rainfall timeseries is stationary, we performed different versions of the augmented Dickey-Fuller test on the data. The results are shown in Table 4.4.7. The null hypothesis was rejected in all

Specification	Statistic	Rainfall
Basic	Z	-6.042
	P	0.000
With trend	Z	-6.066
	P	0.000
With drift	Z	-6.042
	P	0.000

Table 4.4.7: Results of the augmented Dickey–Fuller test for the rainfall time-series in Kianyaga district. The trend specification includes a trend term in the associated regression, and assumes that the process under the null hypothesis is a random walk (possibly with drift). The drift specification assumes that the process under the null hypothesis is a random walk with nonzero drift. Significantly negative test statistics are evidence for stationarity.

cases, suggesting that the rainfall timeseries is stationary.

These results put us in a position to address our main question of interest: does lack of rain predict cortisol levels 10-20 days later? We address this question using a regression of cortisol levels on current and lagged rainfall data. Table 8 reports the results form this regression. The coefficients on the 0th and 1st lag of the rainfall variable are both negative; those on the 1st lag are highly significant. This result suggests that lack of rainfall indeed leads to higher cortisol levels in our respondents. To assess the joint significance of the lagged rainfall coefficients, we performed an F-test; in both speficications, the results were highly significant, confirming the suggestion that the absence of rain indeed leads to raised cortisol levels. In terms of magnitude, we found that a 10mm decrease in rainfall in the previous dekad leads to a cortisol increase of 5.5 nmol/l; from a baseline of 35.4 nmol/l, this corresponds to a 15.5% increase. Thus, the lack of rain in an area of Kikuyu farmers has a strong and temporally contiguous effect on cortisol levels.

4.5 Discussion

In this paper we asked the question whether increases in poverty lead to stress, measured here by the stress hormone cortisol. We find that exogenous increases in poverty indeed lead to stress, in two different settings: first, the proportion of goats and sheep lost to a drought in a sample of 152 Maasai tribespeople in Kajiado district, Kenya, strongly predicts their cortisol levels one year later. We show that the proportion of goats and sheep lost to the

	(1)	(2)
VARIABLES	cort	cort
L0rain	-0.259	-0.257
	(0.209)	(0.210)
L1rain	-0.549***	-0.547***
	(0.142)	(0.142)
female		-2.211
		(6.717)
Constant	41.50***	42.36***
	(4.834)	(5.820)
Observations	283	283
R-squared	0.021	0.021
F-test rain	7.676	7.670
Prob > F	0.000568***	0.000572***

Table 4.4.8: Lack of rain increases cortisol levels in Kianyaga district. Each column shows an OLS regression of baseline cortisol levels, in nmol/l, on current and lagged rainfall, in millimeters. The temporal resolution of the rainfall measures is 10 days (dekadal). The coefficients on the lagged rainfall measures are negative, and the coefficient on the lagged rainfall measure is highly significant. In addition, F-tests for joint significance of the lagged rainfall coefficients confirm that they are jointly different from zero. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

drought is not predicted by observable household characteristics, supporting the claim that the loss of livestock was a random negative shock against which even wealthy households could not protect themselves, and that therefore our findings reflect the causal effect of livestock deaths on cortisol levels. Second, in a sample of 283 Kikuyu farmers in Kinayaga district, Kenya, we combine cortisol measures with household-level GPS data and high-resolution satellite rainfall data and find that lack of rain strongly raises cortisol levels, with a lag of 10-20 days. In both studies, the cortisol increases induced by the negative shocks were significant and large in magnitude. Together, these results suggest that random exogenous increases in households' economic situations – either through livestock deaths, or through weather shocks to agricultural productivity – cause substantial increases in the levels of the stress hormone cortisol.

These results contribute to the emerging literature on the relationship between stress and income/socioeconomic status by showing that this relationship is causal. A growing number of studies have documented that poor and otherwise disadvantaged people show increased levels of cortisol (Cohen et al., 2006a, 2006b; Evans & Kim, 2009; Evans & English, 2002; Li et al., 2007; Lupien et al., 2000; Arnetz et al., 1991; Dowd et al., 2009); however, to date this relationship has been identified through correlation, leaving it unclear in which direction causality runs. One could easily imagine it going in both directions: the idea that poverty can cause stress is uncontroversial; conversely, however, it is also possible that stressed individuals are more likely to end up in poverty, e.g. through impaired job performance due to stress. The contribution of this study is to provide causal evidence for the first channel, i.e. the effect of poverty on stress.

In providing evidence for a causal effect of poverty on stress, our study is similar to two recent studies that have also attempted to resolve this correlation-causation dilemma in the opposite direction, namely by measuring the impacts of development programs on stress levels. Fernald & Gunnar (2009) measured cortisol levels in children who had been exposed to the Mexican PROGRESA program – a comprehensive conditional cash transfer program with a focus on health and education. The authors found that children who had been exposed to the program exhibited lower baseline cortisol levels than those children who had not been in the program. In another study, Fernald et al. (2008) investigated responses to stress and depression questionnaires in a sample of South-African respondents after they were randomly assigned to receive a loan. Those who had received loans showed lower levels of depressive symptoms than the control group; interestingly, however, questionnaire-assessed stress levels were higher after receiving a loan than in the control group, possibly due to the stress induced by having to pay back

the loan at a high interest rate (200% p.a.). Thus, these previous programs studied the effects of poverty decreases on stress levels; note though that one was in children and the other did not measure stress using cortisol. Our study contributes by showing a significant causal effect of poverty on stress in the other direction, namely poverty increases; and by providing cortisol evidence from adults rather than children.

Several caveats are in order. First, in Kajiado district, the possibility remains that unobserved heterogeneity accounts for both the cortisol results and livestock deaths; for instance, pre-existing mental health disorders might lead to both high livestock losses and high levels of cortisol. Indeed, we do observe high levels of depressive symptoms in this sample; ~80% of respondents meet the DSM-IV criteria for severe depression. We are currently gathering GPS location data on all households in this sample, which will make it possible to relate the livestock deaths directly to drought in the region in future work. Nevertheless, this alternative explanation does not fully account for the effects we observe, since

A second potential concern is that respondents who experienced livestock losses now have to perform more strenuous physical activity, and that our cortisol results reflect this physical strain as opposed to psychological stress. However, we deem this account unlikely for two reasons. First, controlling for a Yes/No question about whether the respondent performed strenuous physical activity prior to the interview did not alter the results reported in Tables 3-4. Second, the effect in the Kianyaga sample would be predicted to go in the opposite direction: farmers in this region work harder when it rains compared to when it does not rain – simply put, there is nothing to do without rain. Thus, on this account we should observe an increase in cortisol levels after rain, rather than the decrease that we actually observe.

In addition, the present study raises a number of questions for follow-on work. In particular, we study the effect of exogenous increases in poverty on cortisol levels; it remains unclear whether decreases in cortisol have the converse effect, and could therefore be used as potential stress alleviation interventions. As mentioned above, Fernald et al. (2009) showed that Mexican children whose mothers had been exposed to the Progresa program in Mexico show lower cortisol levels than comparison children; however, it is not clear which of the program's many interventions accounts for this effect, and it remains unknown to what extent selection of mothers into the study could be responsible for it. In addition, data on the effect of poverty alleviation programs on the cortisol levels of adults is not available. We are currently conducting two randomized controlled trials in Kenya, one on health insurance and one on unconditional cash transfers, which will address these issues.

A further remaining question is through which psychological channels

increases or decreases in poverty take their effect on stress. Do people worry more or less about their economic future? Do they socialize less after having been hit by an economic shock, either because they fall from grace or because they have less time given their new economic constraints? It remains an important task for future work to expand on the "closed-form" approach we used here, in which we simply study the relationship between changes in poverty on cortisol levels; what is on people's mind as these economic changes take effect is equally important.

The present study is the first addressing the relationship between poverty and stress in a developing country. This is somewhat surprising as existing data on the prevalence of stress and depression suggest that devleoping countries are particularly affected; we therefore hope that significantly more effort will be dedicated in the future on elucidating the causes and consequences of this fact, and on developing interventions to alleviate the problem.

Bibliography

[1] Aardal, E., & Holm, A. C. (1995). Cortisol in saliva – reference ranges and relation to cortisol in serum. Eur. J. Clin. Chem. Clin. Biochem., 33, 927–932.

[2] Aardal-Ericsson, E., Karlberg, B.E., & Holm, A.C. (1998). Salivary cortisol – an alternative to serum cortisol determinations in dynamic function tests. Clin. Chem. Lab. Med., 36, 215–222.

[3] Akaike, H. (1973). Information theory and an extension of the maximum likelihood principle. In B. N. Petrov & F. Caski (Eds.), Proceedings of the Second International Symposium on Information Theory. Budapest: Akademiai Kiado.

[4] Akaike, H. (1974). A new look at the statistical model identification. IEEE Transactions on Automatic Control, 19, 716-723.

[5] Akaike, H. (1978). On the likelihood of a time series model. The Statistician, 27, 217-235.

[6] Arnetz, B.B., Brenner, S.O., Levi, L., Hjelm, R., Petterson, I.L., Wasserman, J., Petrini, B., Eneroth, P., Kallner, A., Kvetnansky, R., et al. (1991). Neuroendocrine and immunologic effects of unemployment and job insecurity. Psychotherapy and Psychosomatics, 55, 76-80.

[7] Baum A, Garofalo JP, Yali AM. Socioeconomic status and chronic stress: does stress account for SES effects on health? Ann NY Acad Sci 1999;896:131–44.

[8] Bolton, P., Wilk, C.M., Ndogoni, L. (2004). Assessment of depression prevalence in rural Uganda using symptom and function criteria. Soc Psychiatry Psychiatr Epidemiol, 39, 442–447.

[9] Brunner E. Stress and the biology of inequality. Br Med J 1997;314:1472–76.

[10] Cohen S., Doyle, W.J., & Baum, A. (2006a). Socioeconomic status is associated with stress hormones. Psychosom Med, 68, 414-420.

[11] Cohen, S., Schwartz, J., et al. (2006b). Socioeconomic status, race and diurnal cortisol levels in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Psychosom Med, 68, 41-50.

[12] Dowd, J.B., Simanek, A.M., & Aiello, A.E. (2009). Socio-economic status, cortisol and allostatic load: a review of the literature. International Journal of Epidemiology, 38, 1297–1309.

[13] Easterlin, R.A. (1974). Does Economic Growth Improve the Human Lot? In David, P.A., & Reder, M. W. (Eds.), Nations and Households in Economic Growth: Essays in Honor of Moses Abramovitz. New York: Academic Press, Inc.

[14] Elliott, G., Rothenberg, T.J., & Stock, J.H. (1996). Efficient Tests for an Autoregressive Unit Root. Econometrica, 64, 813–836.

[15] Evans, G.W., & English, K. (2002). The environment of poverty: multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. Child Development, 2002, 73, 1238–1248.

[16] Evans, G.W., & Kim, P. (2009). Childhood poverty and health: cumulative risk exposure and stress dysregulation. Psychological Science, 2007, 18, 953–57.

[17] Fernald, L.C.H., & Gunnar, M.R. (2009). Poverty-alleviation program participation and salivary cortisol in very low-income children. Social Science & Medicine, 68, 2180-2189.

[18] Fernald, L.C.H., Hamad, R., Karlan, D., Ozer, E., & Zinman, J. (2008). Small individual loans and mental health: a randomized controlled trial among South African adults. BioMed Central Public Health, 8, 409.

[19] Hammen, C. (2005). Stress and Depression. Annual Review of Clinical Psychology, 1, 293-319.

[20] Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. Neuropsychopharmacology, 23, 477-501.

[21] Kirschbaum, C., & Hellhammer, D.H. (1989). Salivary cortisol in psychobiological research: an overview. Neuropsychobiology, 22, 150–169.

[22] Kristenson M, Eriksen HR, Sluiter JK, Starke D, Ursin H. Psychobiological mechanisms of socioeconomic differences in health. Soc Sci Med 2004;58:1511–22.

[23] Li, L., Power, C., Kelly, S., Kirschbaum, C., & Hertzman, C. Life-time socio-economic position and cortisol patterns in mid-life. Psychoneuroendocrinology, 2007, 32, 824–33.

[24] Lupien SJ, de Leon M, de Santi S et al. Cortisol levels during human aging predict hippocampal atrophy and memory deficits. Nat Neurosci 1998;1:69–73.

[25] Patel, V., Chisholm, D., Rabe-Hesketh, S., Dias-Saxena, F., Andrew, G., & Mann, A. (2003). Efficacy and cost-effectiveness of drug and psychological treatments for common mental disorders in general health care in Goa, India: a randomised, controlled trial. Lancet, 361, 33-39.

[26] Schwarz, G.E. (1978). Estimating the dimension of a model. Annals of Statistics, 6, 461–464.

[27] Sobocki, P., Jönsson, B., Angst, J., & Rehnberg, C. (2006). Cost of depression in Europe. J Ment Health Policy Econ, 8, 87-98.

[28] Steptoe, A., Feldman, P.J., Kunz, S., Owen, N., Willemsen, G.H., Marmot, M., 2002. Stress responsivity and socioeconomic status. A mechanism for increased cardiovascular risk? Euro. Heart J. 23, 1757–1763.

[29] Stevenson, B., & Wolfers, J. (2008). Economic Growth and Subjective Well-Being: Reassessing the Easterlin Paradox. Brookings Papers on Economic Activity.

[30] Thavichachart, N., Intoh, P., Thavichachart, T., Meksupa, O., Tangwongchai, S., Sughondhabirom, A., Worakul, P. (2001) Epidemiological survey of mental disorders and knowledge attitude practice upon mental health among people in Bangkok Metropolis. J Med Ass Thailand, 84, 118–126.

[31] The World Bank (2007). Understanding Poverty.

[32] Van Cauter, E., Leproult, R., & Kupfer, D.J. (1996). Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. J Clin Endocrinol Metab, 81, 2468 –73.

Chapter 5

The Psychology of Poverty

5.1 Summary

Does poverty have psychological consequences? Beginning with Easterlin (1974), a growing literature has investigated the association between income levels on the one hand, and psychological outcomes on the other. However, this literature remains equivocal on whether a relationship exists; in addition, it focuses almost exclusively on happiness and life satisfaction, while little evidence exists about the link between poverty and other psychological outcomes. Using World Value Survey data from 58,685 respondents in 41 countries, we show here that poverty is associated with a host of psychological outcomes other than happiness and life satisfaction. Compared to the rich, the poor have more internal locus of control, less intrinsic motivation, less prosocial attitudes, feel more lonely, and show more symptoms of depression. These findings hold both for poor compared to rich people in the same country, and for poor compared to rich countries. The results remain robust after controlling for socioeconomic variables, and do not depend on whether income is coded linearly or as the logarithm. Across countries, income level is more important than income growth in determining psychological outcomes. In addition, we use coastal access as an instrument for country-level GDP and find evidence for the causality of the link from income to locus of control using this strategy. Together, our results suggest that poverty may have broad psychological consequences, and that some of these links are causal.

5.2 Introduction

Poverty is most frequently described in terms of material scarcity. However, a small number of scholars have suggested over the centuries that the lives

of the poor may be characterized not only by material deprivation, but also negative psychological outcomes. In the Theory of Moral Sentiments, Adam Smith cast poverty in terms of its psychological consequences: "[t]he reason poverty causes pain is not just because it can leave people feeling hungry, cold and sick, but because it is associated with unfavourable regard. ... The poor man ... is ashamed of his poverty." In his prominent ethnography Five Families: Mexian Case Studies in the Culture of Poverty (1959), anthropologist Oscar Lewis argued that the lives of the poor were characterized by "a strong feeling of marginality, of helplessness, of dependency, of not belonging ... of inferiority, of personal unworthiness." A few years later, the controversial Moynihan report claimed that Black (poor) families in the United States were caught in a "tangle of pathology" (Moynihan, 1965).

These suggestions are easy to misunderstand as blaming the poor for their poverty by attributing to them certain psychological "shortcomings" which keep them mired in poverty. Possibly for this reason, the suggestion that poverty may have adverse psychological consequences has only recently received attention in the literature. In particular, it was not until the 1970s that the obvious question that follows from this assertion was posed in earnest: is it true empirically that poverty has psychological consequences (or at least correlates)?

Most of the resulting literature has concerned itself with the empirical relationship between income and self-reported happiness. Are poor people unhappier than richer people? In a series of papers, Richard Easterlin found that happiness and income were correlated within countries, but not across countries; in particular, increases in national income over time did not translate into increases in average self-reported happiness. Within countries, however, the poor consistently reported lower levels of happiness than the rich (Easterlin, 1974, 1995, 2001, 2003; Easterlin & Angelescu, 2009).

More recently, however, Easterlin's cross-country finding has been convincingly overturned by an analysis of a large number of datasets from a broad panel of countries by Betsey Stevenson and Justin Wolfers (2008). These authors find a robust relationship between self-reported happiness and life satisfaction both within and across countries.

Thus, it appears that poverty indeed is associated with self-reported happiness and life satisfaction, both within and across countries. However, it remains unclear whether poverty affects psychological outcomes beyond self-reported happiness and life satisfaction. In this paper, we use World Value Survey data from 58,685 respondents in 41 countries and show that both within and across countries, poor people have more internal locus of control, less intrinsic motivation, less prosocial attitudes, feel more lonely, and show more symptoms of depression than richer people.

A further important question regarding the relationship between income and psychological outcomes is its causality. One can easily see how poverty can both be a cause and a consequence of certain psychological outcomes; e.g., depression may lead to unemployment, and unemployment may in turn exacerbate depression. In addition, third factors such as culture or religion may affect both income and psychological outcomes simultaneously. It is important both for scientific and policy purposes to elucidate the directionality and magnitude of the causal realtionships between income and psychological outcomes. The reverse and simultaneous causation biases just described complicate this undertaking. However, they can be overcome using exogenous influences on one of the variables of interest. In the ideal case, this is achieved through randomized experiments; in the context of income and psychological outcomes, for instance, one could randomly assign cash transfers to some households but not others, and thus identify the causal effect of a change in income on psychological outcomes (we are currently conducting such experiments in Western Kenya and Nairobi). Across countries, however, this approach is not feasible; for a host of reasons one cannot randomly assign income shocks to some countries but not others in order to measure their effect on psychological outcomes. However, natural experiments offer a window onto the causality of the relationship between income and psychological outcomes even across countries: if, for instance, some countries happen to have particular geographical features that affect only income but are unlikely to affect psychological outcomes through channels other than income, instrumental variables regression can be used to isolate the causal effect of income on psychological outcomes. In this paper we employ this strategy to address the causality question. Specifically, we use the proportion of a country's land area within 100km from the coast, and the poportion of the population living in this area, as instruments for income. It has long been known that coast access is a crucial determinant of a country's income, mainly because it gives the country's economy access to trade routes and foreign markets (Gallup et al., 1999). On the other hand, we argue that it is unlikely that coast access affects psychological outcomes through channels other than income (see Methods and Discussion for details). Using this strategy, we find a causal effect of income on locus of control across countries: higher income, instrumented by coastal access, causes more internal locus of control.

Together, our results suggest that poverty has broad psychological consequences, and that causality runs from income to psychology in at least some cases. We suggest that these psychological outcomes could serve as welfare indicators against which development policies are evaluated; moreover, to the extent that these psychological variables themselves influence income levels, they could become promising targets for development interventions.

5.3 Data and Empirical Strategy

5.3.1 Data sources

5.3.1.1 Psychological variables

The psychological outcome variables were taken from the World Value Survey and the European Value Survey (www.wvs.org). For our variables of interest, the data cover 58,685 respondents in 41 countries. Most of the country-specific samples are nationally representative; we exlude countries for which the national samples are not representative or for which sampling weights are not available. The World Value Survey is administered in discrete waves; some of our variables were collected in all waves, while others were collected only in some waves (see Table 5.3.1). Altogether, our data cover the years 1980-2008. In all regressions, we use year fixed effects to account for any possible time trends.

Table 5.3.1 summarizes the psychological variables we tested. They were chosen to tap into the following constructs: locus of control, intrinsic motivation, prosocial attitudes, loneliness, and depression symptoms (meaninglessness). These constructs were chosen because they are both economically important and relevant for psychological well-being and welfare.

5.3.1.2 Within-country income data

Within-country income data was also obtained from the World Value Survey and European Value Survey data. Specifically, the questionnaires for all countries countained a variable asking respondents about their household income. This variable was coded on a scale from 1-10, corresponding to income deciles for the country of residence. Thus, 1 means than the respondent was in the lowest 10% of income earners in their country of residence; 10 indicates that they were in the top 10%. We use this variable as the main independent variable in the within-country regressions.

5.3.1.3 GDP and growth data

GDP and GDP growth data were taken from the World Development Indicators published by the World Bank. The data cover the years 1960-2010. GDP is measured per capita at purchasing power parity in constant 2005 US dollars. Growth is measured in % of real GDP. These variables are the main independent variables in the cross-country regressions.

Variable name	Construct measured	Survey question	Waves	Number of respondents	Number of countries
shapefateyrself	Locus of control	Some people believe that individuals can decide their own destiny, while others think that it is impossible to escape a predetermined fate. Please tell me which comes closest to your view on this scale on which 1 means "everything in life is determined by fate," and 10 means that "people shape their fate themselves."	2005-2008	65,958	48
intrinsic	Intrinsic motivation	I do the best I can regardless of pay (0=no, 1=yes)	1989-1993	52,560	38
helpneighborhood	Prosocial attitudes	Would you be prepared to actually do something to improve the conditions of people in your neighbourhood/community? (1=absolutely no ... 5=absolutely yes)	1999-2004	37,940	31
helpfamily	Prosocial attitudes	Would you be prepared to actually do something to improve the conditions of your immediate family? (1=absolutely no ... 5=absolutely yes)	1999-2004	38,179	31
lonely	Loneliness	Do you ever feel very lonely? (1=never ... 4=frequently)	1981-1984	19,129	16
lifemeaningless	Depression symptoms	How often, if at all, do you have the feeling that life is meaningless? (1=never ... 4=often)	1981-1984	18,965	16

Table 5.3.1: Psychological variables of interest.

5.3.1.4 Geographical data

For the instrumental variables regression, we obtained country-level data on coastal access from the Center for International Development at Harvard University (http://www.cid.harvard.edu/ciddata/geographydata.htm#general). The specific variables we use are a) the proportion of land area of the country that is within 100km from an icefree coast (lc100km), and the proportion of the population that lives within 100km from an icefree coast (pop100km).

5.3.2 Model specifications

5.3.2.1 Within-country regressions

Our first question is whether within countries, poorer respondents show different psychological outcomes compared to richer respondents. Do poorer people differ in locus of control, different intrinsic motivation, prosocial attitudes, loneliness, or meaninglessness? To answer these questions, we regress each variable of interest on the within-country income categories for each respondent household. Specifically, we fit the following OLS model for each variable of interest:

$$Psych_{i,c,t} = \alpha_c + \mu_t + \beta Inc_{i,c,t} + \gamma_{i,c,t}\mathbf{X}_{i,c,t} + u_{i,c,t},$$

where $Psych_{i,c,t}$ is the household-, country-, and year-specific response on the psychological variable of interest, $Inc_{i,c,t}$ is the household's income category on a scale from 1 (low) to 10 (high), coded either linearly or as the logarithm (see Results), the coefficients α_c and μ_t represent country and year fixed effects, respectively, and $u_{c,t}$ is the error term. $\mathbf{X}_{i,c,t}$ is a vector of controls that includes dummy variables for gender and marital status, as well as education (in years), number of children, a quartic in age, and the quartic in age interacted with gender.

5.3.2.2 Cross-country regressions

Cross-section: Across countries, we first obtained aggregate indices for each of the psychological variables by fitting an ordered probit model of the form

$$Psych_{c,t,} = \gamma_c + \delta_t + \varepsilon_{c,t},$$

where $Psych_{c,t}$ is the country- and year-specific aggregate response on the psychological variables of interest, γ_c and δ_t are country and year fixed effects, and $\varepsilon_{c,t}$ is the error term. We use the sampling weights provided in the

WVS data. This regression generates predicted values for the psychoogical variables net of country and year fixed effects. We then fit the following ordinary least squares model to the resulting country- and year-specific aggregate probit index:

$$\widehat{Psych}_{c,t} = \alpha_c + \mu_t + \beta Inc_{c,t} + \mu_{c,t},$$

where $\widehat{Psych}_{c,t}$ is the probit index of the psychological outcome variable in question for country c in year t, $Inc_{i,c,t}$ is either GDP, log GDP, GDP growth, or a combination of two of these variables, for country c in year t. The coefficients α_c and μ_t represent country and year fixed effects, respectively, and $u_{c,t}$ is the error term. Standard errors are clustered by country.

Instrumental variables: For the first stage of the instrumental variables regression of psychological outcomes on income, when income is instrumented by coastal access, we regress income on coastal access using the following OLS specification:

$$Inc_{c,t} = \alpha_c + \mu_t + \theta_1 pop100km_c + \theta_2 lc100km_c + u_{c,t},$$

where the variables are as described above. In the second stage, we regress psychological outomces – in particular, locus of control – on the predicted income variable from the first-stage regression:

$$Psych_{c,t} = \alpha_c + \mu_t + \beta \widehat{Inc}_{c,t} + u_{c,t},$$

where $\widehat{Inc}_{c,t}$ represents the income variable instrumented by coastal access, i.e. free from reverse and simultaneous causality bias.

5.4 Results

5.4.1 Within-country results

Figure 5.4.1 plots responses to the psychological survey questions against within-country income category. The corresponding regression results are shown in Table 5.4.1. Each column represents one psychological outcome variable, corresponding to Table 5.3.1; the rows represent the regression coefficients on the predictor variables and their significance levels. The crucial row for our purposes is that showing the coefficients on the income variable. It can be seen that all of these coefficients are different from zero and highly significant. Specifically, poorer individuals show more internal locus

of control, less intrinsic motivation, less prosocial attitudes, more feelings of loneliness, and more symptoms of depression than richer individuals. These results are robust to the inclusion of control variables for gender, marital status, education, number of children, a quartic in age, and the quartic in age interacted with gender, and to controlling for country- and year fixed effects. Thus, this table reveals a robust relationship between the six psychological outcomes of interest and the household's income.

5.4.2 Cross-country results

We next asked whether the relationship between income and psychological outcomes that we observe within countries is also evident across countries. In other words, do richer countries have, on average, more intrinsic locus of control, etc.? In addition, to countries that grow fast than others show differences on any of these variables?

Figure 5.4.2 illustrates the relationship between psychological outcomes and GDP across countries, and Figure 5.4.3 illustrates the relationship between psychological outcomes and GDP growth across countries. The corresponding cross-country regression results are shown in Table 5.4.2, where different subsections show results for the regressions on GDP levels, growth, or both, and expressing levels and growth rates either in linear or log terms. As can be seen from Figure 5.4.2 and Table 5.4.2, poorer countries show more internal locus of control, less intrinsic motivation, less prosocial attitudes, more feelings of loneliness, and more symptoms of depression than richer countries. These results are all statistically significant in at least one specification, and identical in sign compared to the within-country results. In contrast, however, the growth results are overwhelmingly non-significant. This suggests that people in richer countries have, on average, more internal locus of control, more intrinsic motivation, more prosocial attitudes, less loneliness, and fewer feelings of meaninglessness than people in poorer countries. However, the same is not true for countries that grow at higher vs. lower rates; GDP growth is not significantly related to any of these variables.

To address the question of causality in the relationship between income across countries and psychological outcomes, we next used instrumental variables regression to ask whether income has a causal effect on psychological outcomes when it is instrumented using coastal access. We focus on locus of control as an outcome variable, since we have 41 datapoints (countries) for this variable, while the other variables are only available in fewer countries.

Two criteria need to be fulfilled for the validity of coastal access as an instrument. The first is the exclusion restriction: the instrument should not affect the outcome variable (in our case, psychological outcomes) through

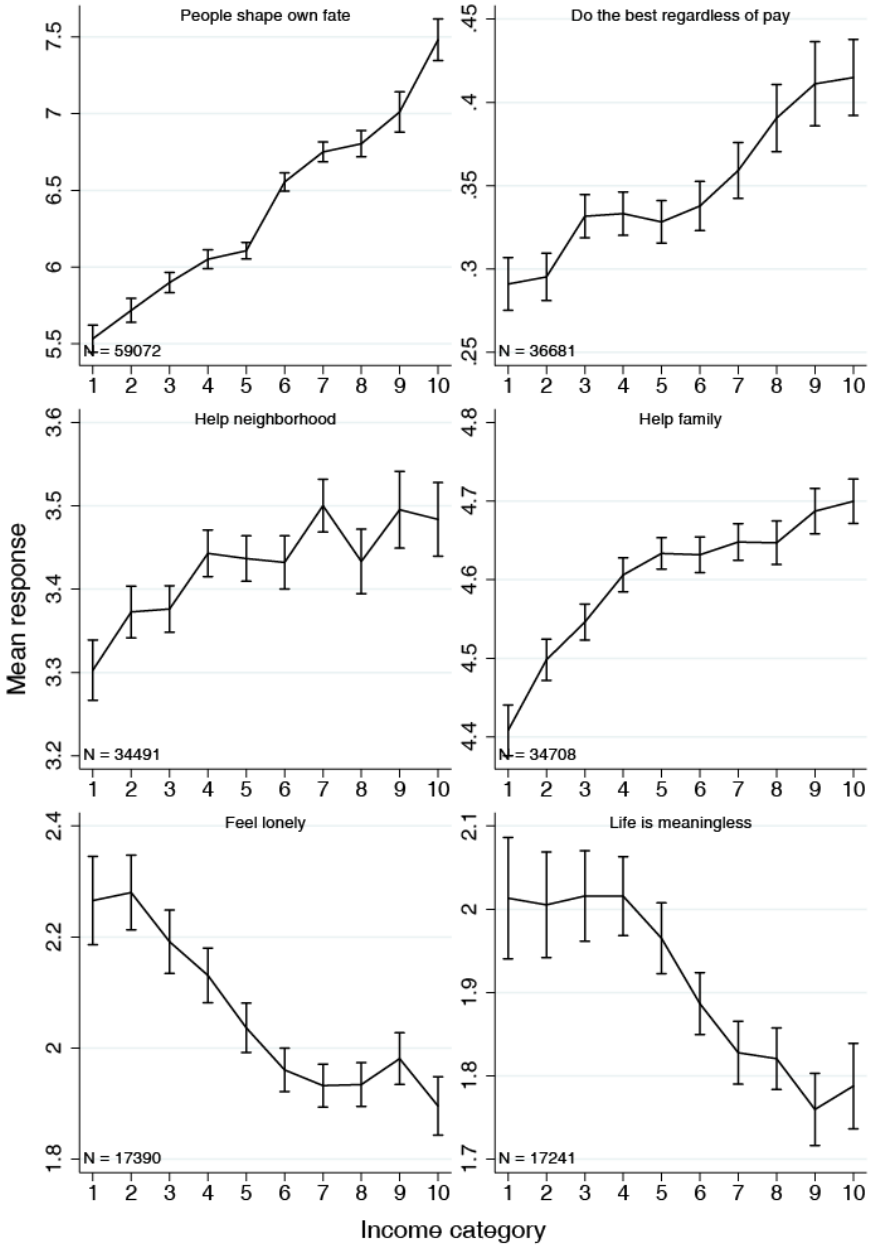

Figure 5.4.1: Psychological variables against income category within countries. The lines represent the mean response to each psychological survey question for the particular income category (from 1-10), collapsed across countries. Error bars represent one standard error of the mean.

VARIABLES	(1) People shape own fate	(2) Do the best regardless of pay	(3) Help neighborhood	(4) Help family	(5) Feel lonely	(6) Life is meaningless
Income (steps 1-10, log)	0.359***	0.0211	0.0220**	0.0583***	-0.144***	-0.126***
	(15.28)	(1.193)	(2.200)	(7.252)	(-7.871)	(-6.903)
Female	0.446	-1.600	0.372	0.621	0.897	0.741
	(0.437)	(-0.335)	(0.795)	(1.582)	(1.445)	(1.085)
Education (years)	0.131***	0.0164**	0.0186***	0.0202***		
	(21.37)	(2.433)	(6.440)	(9.027)		
Married	0.0888***	0.000904	0.0992***	0.119***	-0.502***	-0.193***
	(3.040)	(0.0407)	(7.563)	(11.11)	(-20.86)	(-8.269)
Number of children	-0.027***	-0.00233	0.0134***	0.0240***	-0.00160	-0.00145
	(-3.170)	(-0.576)	(3.193)	(7.130)	(-0.424)	(-0.395)
Age	-0.0808	0.00681	-0.0641**	-0.0131	0.0891**	-0.0435
	(-1.101)	(0.0170)	(-2.077)	(-0.515)	(2.062)	(-0.915)
Age^2	0.00240	-0.000671	0.00238**	0.000527	-0.00221	0.00159
	(0.973)	(-0.0390)	(2.375)	(0.634)	(-1.526)	(0.995)
Age^3	-3.19e-05	1.81e-05	-3.28e-05**	-8.93e-06	2.01e-05	-2.38e-05
	(-0.923)	(0.0576)	(-2.407)	(-0.784)	(0.992)	(-1.059)
Age^4	1.53e-07	-1.52e-07	1.52e-07**	4.72e-08	-4.72e-08	1.22e-07
	(0.889)	(-0.0731)	(2.309)	(0.850)	(-0.469)	(1.087)
Female x Age	-0.0781	0.219	-0.0255	-0.0432	-0.0334	-0.0526
	(-0.793)	(0.384)	(-0.580)	(-1.151)	(-0.560)	(-0.792)
Female x Age^2	0.00304	-0.0103	0.000612	0.000899	0.000373	0.00157
	(0.915)	(-0.424)	(0.425)	(0.717)	(0.188)	(0.698)
Female x Age^3	-4.77e-05	0.000203	-4.78e-06	-4.77e-06	4.02e-06	-1.86e-05
	(-1.024)	(0.454)	(-0.243)	(-0.272)	(0.146)	(-0.589)
Female x Age^4	2.52e-07	-1.41e-06	3.04e-09	-1.32e-08	-6.25e-08	7.04e-08
	(1.083)	(-0.475)	(0.0317)	(-0.151)	(-0.463)	(0.446)
Constant	5.863***	1.700	3.266***	2.993***	0.297	1.843*
	(3.515)	(0.226)	(4.424)	(4.894)	(0.302)	(1.699)
Observations	58685	1677	31072	31258	15823	15703
R-squared	0.175	0.016	0.184	0.137	0.143	0.050
Country FE	YES	YES	YES	YES	YES	YES
Year FE	YES	YES	YES	YES	YES	YES

Table 5.4.1: Within-country regression results. Each column shows the effect of within-country income category (from 1-10) and control variables on one psychological variable of interest. All models include country and year fixed effects. Robust t-statistics in parentheses. *** p<0.01, ** p<0.05, * p<0.1

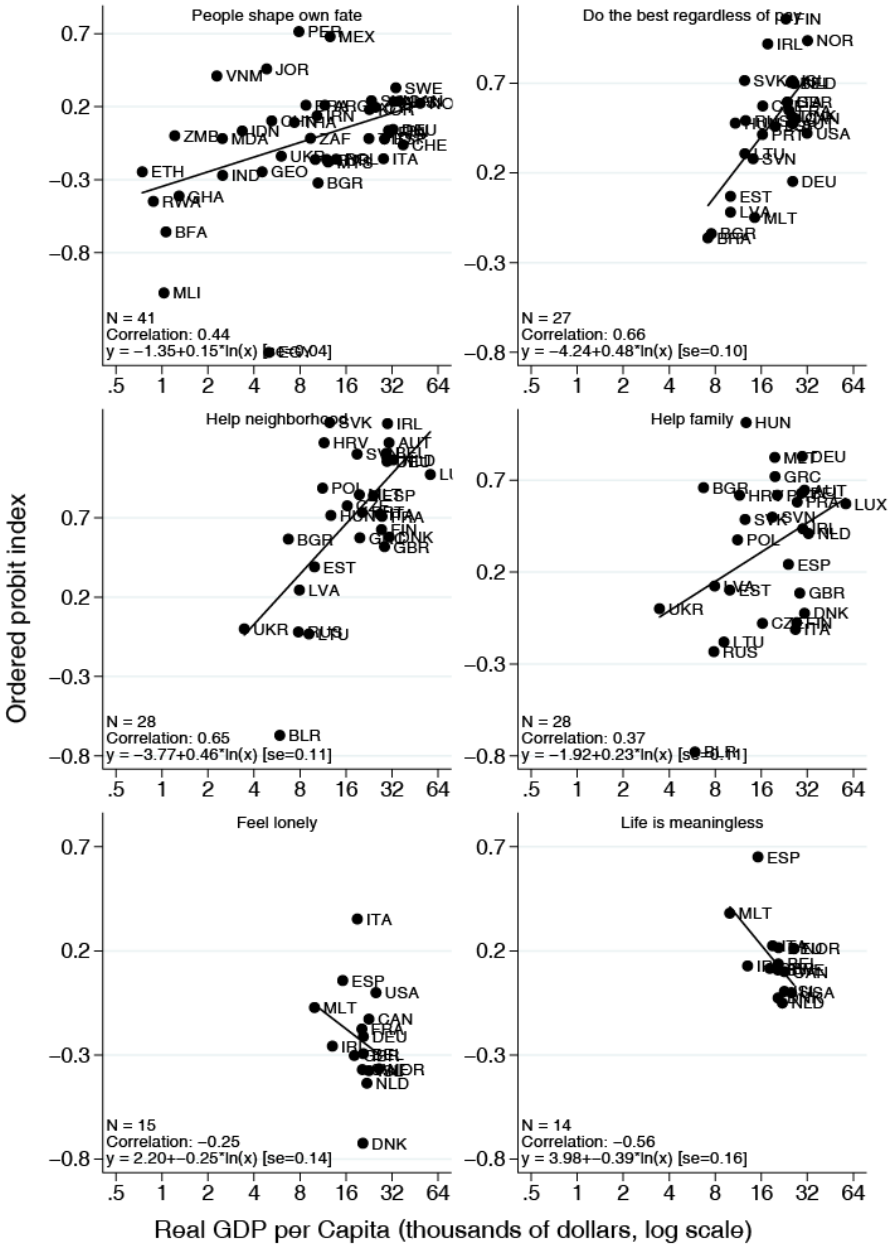

Figure 5.4.2: Cross-country scatterplots showing psychological outcomes against GDP.

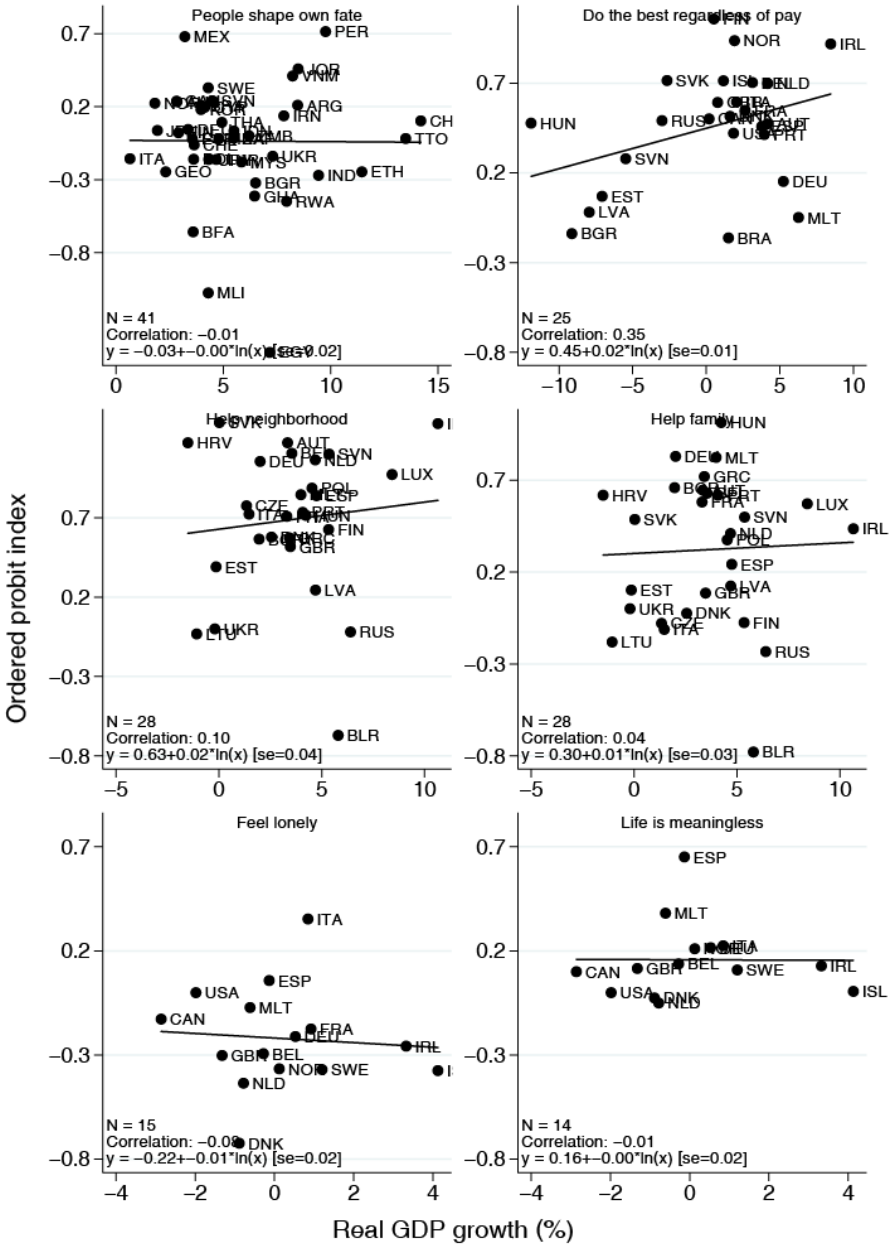

Figure 5.4.3: Cross-country scatterplots showing psychological outcomes against growth.

	(1) People shape own fate	(2) Do the best regardless of pay	(3) Help neighborhood	(4) Help family	(5) Feel lonely	(6) Life is meaningless
Model 1: GDP (log)						
GDP (log)	0.145***	0.480***	0.458***	0.230*	-0.246	-0.388**
	(3.527)	(4.798)	(4.096)	(2.011)	(-1.724)	(-2.415)
Constant	-1.350***	-4.244***	-3.767***	-1.921	2.203	3.981**
	(-3.321)	(-4.335)	(-3.362)	(-1.684)	(1.583)	(2.483)
R-squared	0.190	0.431	0.423	0.136	0.063	0.311
Model 2: GDP						
GDP	1.03e-05***	2.57e-05***	2.08e-05**	1.02e-05*	-1.44e-05	-2.28e-05*
	(2.712)	(3.718)	(2.655)	(1.745)	(-1.527)	(-2.141)
Constant	-0.187*	-0.0435	0.265	0.115	0.0652	0.607**
	(-1.728)	(-0.294)	(1.336)	(0.695)	(0.344)	(2.639)
R-squared	0.112	0.360	0.282	0.085	0.063	0.309
Model 3: GDP growth						
GDP growth	-0.000913	0.0225	0.0171	0.00576	-0.0110	-0.000620
	(-0.0567)	(1.705)	(0.435)	(0.228)	(-0.489)	(-0.0337)
Constant	-0.0309	0.449***	0.629***	0.301***	-0.218***	0.157***
	(-0.316)	(7.141)	(4.112)	(2.811)	(-3.250)	(3.060)
R-squared	0.000	0.121	0.010	0.001	0.007	0.000
Model 4: GDP & GDP growth						
GDP	1.37e-05***	2.66e-05**	2.39e-05**	1.20e-05	-1.59e-05*	-2.38e-05*
	(2.904)	(2.494)	(2.689)	(1.464)	(-2.067)	(-2.178)
GDP growth	0.0287	-0.000611	-0.0294	-0.0176	-0.0183	-0.0119
	(1.383)	(-0.0349)	(-0.665)	(-0.525)	(-0.817)	(-0.815)
Constant	-0.398**	-0.0697	0.304	0.138	0.0977	0.628**
	(-2.069)	(-0.304)	(1.562)	(0.938)	(0.603)	(2.677)
R-squared	0.147	0.367	0.306	0.096	0.080	0.324
Model 5: GDP (log) & GDP growth						
GDP per capita (log)	0.178***	0.532***	0.508***	0.261*	-0.267**	-0.401**
	(3.741)	(3.431)	(4.061)	(1.800)	(-2.221)	(-2.450)
GDP growth (%)	0.0296	-0.00660	-0.0306	-0.0188	-0.0173	-0.0106
	(1.507)	(-0.396)	(-0.806)	(-0.629)	(-0.769)	(-0.735)
Constant	-1.810***	-4.758***	-4.150***	-2.156	2.414*	4.116**
	(-3.477)	(-3.112)	(-3.536)	(-1.592)	(2.061)	(2.517)
R-squared	0.231	0.440	0.450	0.149	0.079	0.322
All models:						
Observations	41	25	28	28	15	14
Country FE	YES	YES	YES	YES	YES	YES
Year FE	YES	YES	YES	YES	YES	YES
Clustering by country	YES	YES	YES	YES	YES	YES

Table 5.4.2: Cross-country regression results. Each model shows the effect of GDP, log GDP, or growth, and their combinations, on the psychological variables of interest. All models include country and year fixed effects. Standard errors are clustered by country. Robust t-statistics in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

other channels than the endogenous variable (in our case, income). In our view, it is very plausible that coastal access affects locus of control only through the income channel. In particular, it is widely known that coastal access is a strong predictor of trade openness and access to foreign markets; it ths directly contributes to the economic success of a country. On the other hand, it is more difficult to imagine how it would affect psychological outcomes through channels other than income. We can only think of one candidate channel, i.e. religion: it may be the case that countries with easy coastal access are more likely than other to get missionarized. However, in this case we would likely observe the opposite effect than the one we actually see: in all likelihood the effect of religiosity on locus of control is, if anything, to make it more internal; thus we would expect these countries to show more internal locus of control than others. However, we actually observe the opposite, as will be detailed below. Thus, in our view the coastal access instrument satisfies the exclusion restriction.

The second criterion for instrument validity is relvance: the instrument has to significantly predict the endogenous variable, in this case, GDP. This is tested in the first-stage regression, the results of which are shown in Table 5.4.3. The rule of thumb for instrument relevance is an F-statistic greater than 10 in the first-stage regression. Indeed, we obtain F-statistics greater than 10 in all specifications, whether GDP is specified in linear or log terms, and whether pop100km or lc100km or both variables are used as the instruments. Thus, the instruments are clearly relevant.

We are now in a position to assess the causal effect of GDP on locus of control, using the version of GDP that is instrumented with coastal access. We estimate this model using two-stage least squares (2SLS); the results are shown in Table 5.4.4. It can be seen that all effects are positive and weakly significant; thus, to the extent we trust the validity and exogeneity of our instrment, we conclude that higher incomes cause more internal locus of control.

5.5 Discussion

The purpose of this paper was to ask whether poverty has particular psychological consequences. A long-standing literature has investigated the association between income and happiness; the most recent contributions to this literature document an assocation between income and happiness that obtains both within countries, as well as across countries. Our findings add to this literature in three ways:

First, we extend previous findings to other psychological outcome vari-

VARIABLES	(1) gdp	(2) gdp	(3) gdp	(4) lgdp	(5) lgdp	(6) lgdp
pop100km	10,334***		10,891**	1.227***		1.178**
	(2,233)		(4,953)	(0.243)		(0.539)
lc100km		9,378***	-644.3		1.141***	0.0570
		(2,332)	(5,109)		(0.254)	(0.556)
Constant	10,896***	12,130***	10,881***	8.678***	8.814***	8.679***
	(1,267)	(1,154)	(1,276)	(0.138)	(0.126)	(0.139)
Observations	203	203	203	203	203	203
R-squared	0.096	0.074	0.096	0.112	0.091	0.112
F-value	21.41	16.17	10.66	25.47	20.20	12.68

Table 5.4.3: Instrumental variable regressions: first stage. Each column represents an OLS regression of GDP – either linear or log – on a coastal access variable: either proportion of the population living within 100km from an icefree coast (pop100km), or proportion of land area within 100km from an icefree coast (lc100km), or both. Standard errors in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

	(1) model1	(2) model3	(3) model5	(4) model2	(5) model4	(6) model6
VARIABLES	shapefateyrself_hat	shapefateyrself_hat	shapefateyrself_hat	shapefateyrself_hat	shapefateyrself_hat	shapefateyrself_hat
gdp	1.65e-05*	1.73e-05*	1.65e-05*			
	(9.57e-06)	(9.60e-06)	(9.55e-06)			
lgdp				0.151*	0.148**	0.150**
				(0.0776)	(0.0718)	(0.0727)
Constant	-0.278*	-0.291	-0.278*	-1.399*	-1.379**	-1.394**
	(0.163)	(0.174)	(0.163)	(0.709)	(0.674)	(0.670)
Observations	41	41	41	41	41	41
R-squared	0.072	0.060	0.072	0.190	0.190	0.190
Instrument	pop100km	lc100km	Both	pop100km	lc100km	Both

Table 5.4.4: Instrumental variable regressions: two-stages least squares (2SLS). GDP (linear or log) is instrmented with pop100km or lc100km or both variables. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

ables. For decades, the literature on income and psychological outcomes has mainly been concerned with happiness and life satisfaction; however, data for a host of other important psychological outcome variables is available, and our results show that these variables, too, show strong associations with income. In demonstrating the associations described above, our study lends support to a small number of studies that have shown similar associations for optimism, locus of control, and self-esteem (Maqsud & Rouhani, 1997; Sherman & Hofmann, 1978; Twenge & Campbell, 2002; Scheier & Carver, 1985; Robb et al., 2009; Lynch et al., 1997; Taylor & Seeman, 1999). However, note that these findings were largely obtained in much smaller samples, often in particular population subgroups, and without attempts to establish causal relationships.

Second, our findings lend support to the most recent results by Stevenson & Wolfers, which show that the correlation between income and happiness also holds across countries, in contrast to the early findings by Easterlin (1974). In particular, we find that for all variables under consideration, there is both a within-country correlation with income category, and a cross-country correlation with country GDP. In contrast, however, we find no association between any of our psychological outcome variables and GDP growth; thus, as countries get richer, they do not appear to cahnge much in the psychological variables we consider here. A potential explanation for this somewhat surprising result is that yearly GDP growth, which we used here as the growth variable, may be too volatile and short-term to be reflected in psychological outcomes; a variable that aggregates growth over a longer time horizon may show more of an association with psychological outcomes in future work.

Finally, our findings add to the literature on the relationship between income and psychological variables in general by using coastal access as an instrument, and by showing that income in fact has a causal effect on some psychological outcomes. In our view this approach is superior to other instrumental variable approaches used in this literature. Specifically, Stevenson & Wolfers instrumented income using country and education as income predictors; these instruments are likely to fail the exclusion restriction – it is easy to imagine that both country and education might affect psychological variables in other ways than through income. The instrumental variable we use, coastal access, appears to us to be a much more promising candidate for a good instrument.

In sum, we show a robust effect of income on a variety of psychological outcomes. This relationship is robust to a number of control variables and to country and year fixed effects. It holds both within and across countries, and obtains in the cross-country specifications for GDP levels, but not GDP

growth. Finally, insturmental variable estimates suggest that the relationship between high income and internal locus of control is causal. Together, our findings suggest that poverty may have significant psychological costs that go beyond economic welfare. In our view these costs should be on the radar of policymakers, since they imply that poverty alleviation programs, to the extent that they affect these measures of psychological welfare, have to be evaluated not only in terms of their economic effects, but also on their contributions to psychological well-being.

Bibliography

[1] Easterlin, R.A. (1974). Does Economic Growth Improve the Human Lot? In David, P.A., & Reder, M. W. (Eds.), Nations and Households in Economic Growth: Essays in Honor of Moses Abramovitz. New York: Academic Press, Inc.

[2] Easterlin, R.A. (1995). Will Raising the Incomes of All Increase the Happiness of All? Journal of Economic Behavior and Organization, 27, 35-48.

[3] Easterlin, R.A. (2001). Income and Happiness: Towards a Unified Theory. The Economic Journal, 111, 465-484.

[4] Easterlin, R.A. (2003). Explaining Happiness. PNAS, 100, 11176-11183.

[5] Easterlin, R.A., & Angelescu, L. (2009). Happiness and Growth the World Over: Time Series Evidence on the Happiness-Income Paradox. IZA Discussion Paper No. 4060.

[6] Gallup, J.L., Sachs, J.D., & Mellinger, A.D. (1999). Geography and Economic Development. International Regional Science Review, 22, 179-232.

[7] Lewis, I. (1959). Five Families: Mexian Case Studies in the Culture of Poverty (1959). New York: Basic Books.

[8] Lynch, J. W., Kaplan, G. A., & Shema, S. J. (1997). Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. N Engl J Med, 337, 1889-1895.

[9] Maqsud, M., & Rouhani, S. (1991). Relationships between socioeconomic status, locus of control, self-concept, and academic achievement of batswana adolescents. Journal of Youth and Adolescence, 20, 107-114.

[10] Moynihan (1965). The Negro Family: The Case For National Action. Office of Policy Planning and Research, United States Department of Labor.

[11] Robb, K. A., Simon, A. E., & Wardle, J. (2009). Socioeconomic Disparities in Optimism and Pessimism. Internation Journal of Behavioral Medicine, 16, 331-338.

[12] Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: assessment and implications of generalized outcome expectancies. Health Psychol, 4, 219-247.

[13] Sherman, L., & Hofmann, R. J. (1978). Interrelationship amongst locus of control, socioeconomic status, sex and achievment. Paper presented at the Annual Convention of the American Psycological Association. Toronto, Ontario, Canada.

[14] Smith, A. (1759/2010). The Theory of Moral Sentiments. New York: Penguin Classics.

[15] Stevenson, B., & Wolfers, J. (2008). Economic Growth and Subjective Well-Being: Reassessing the Easterlin Paradox. Brookings Papers on Economic Activity.

[16] Taylor, S. E., & Seeman, T. E. (1999). Psycosocial resources and SES-health relationship. Ann NY Acad Sci, 896, 210-225.

[17] Twenge, J. M., & Campbell, K. W. (2002). Self-Esteem and Socioeconomic Status: A Meta-Analytic Review. Personality and social psychology review, 6, 59-71.

Chapter 6

Negative Income Shocks Increase Present Bias in Intertemporal Choice

6.1 Summary

Poor individuals often exhibit more present-focused time preferences than richer individuals, and negative income shocks have been linked to increases in present-biased intertemporal choice. However, it remains unknown whether this effect is causal, and whether is due to beliefs or preferences: increases in present focus in poverty or after negative income shocks could either be due to more severe environmental constraints in contexts of poverty, or to direct effects of poverty or income shocks on preferences. Here we address these questions in a laboratory experiment in which subjects receive different starting endowments which create "rich" and "poor" groups; in addition, all participants then perform a real effort task to earn money, following which subgroups of participants receive positive and negative income shocks. Subsequently we measure time preferences. We find that negative income shocks, but not positive income shocks, lead to an increase in present bias during intertemporal choice. In contrast, positive income shocks have no such effect; similarly, time preferences are not affected by mean levels of wealth ("rich" vs. "poor"). The effect of negative income shocks on time preference is specific to present bias and does not affect impatience per se. Furthermore, no effects were found on reservation wages. Together, these findings suggest that negative income shocks have a direct causal effect on preferences that is not explained by wealth levels and individual characteristics.

6.2 Introduction

Both empirically and theoretically, poverty has been linked to present-focused time preferences (see literature review below). However, it remains unknown to what extent this relationship is causal; in addition, it is unknown whether an effect of poverty on time preferences is due to beliefs about environmental constraints, or changes in pure preferences. Evidence exists for both views. In favor of the former hypothesis, it has frequently been argued that high rates of time preference may be entirely rational in conditions of poverty – e.g., Becker & Mulligan (1997) show how existing economic conditions such as wealth, and environmental influences such as mortality and risk, can endogenously lead to behavior that looks like impatience. Others have argued that market imperfections, e.g. in insurance and credit markets, are responsible for behavior that resembles high rates of time preference in poor countries, since they may imply that investments with immediate fixed costs and delayed benefits are less attractive than immediate-return investments (Banerjee, 2001; Holden et al., 1998; Pagiola, 1996, Shiferaw & Holden, 2001). Similarly, present-focused decisions may be rational if returns to investment are low, which has also been argued to be the case in developing countries (Rosenzweig, 1995; van Walle, 2003). Finally, present-focused behavior among the poor may occur if people are severely calorie-constrained and have to focus on eating sufficiently today in order to survive until tomorrow (Murphree, 1993; Bardhan, 1996; Lumley, 1997; Dasgupta, 1997). Endogenous time preferences may have significant long-run consequences: Strulik (2011) argued that such endogenous time preferences can account for the growth paths taken by many countries better than the standard Ramsey model, and Chakrabarty (2011) suggested that they can create poverty traps.

However, it could also be the case that conditions of poverty also affect time preferences directly, above and beyond a rational response to environmental conditions. A number of authors have suggested that environments of poverty may have direct impacts on preferences (Bertrand et al., 2004; Hall, 2008). Here we present evidence for this view; specifically, we show that negative income shocks – a salient feature of the lives of many of the world's poorest people – increase present bias in intertemporal choice. Because of the difficulty of studying income shocks while holding constant wealth, this paper takes a laboratory approach, which allows to study the effect of shocks while holding constant individual wealth by a) random assignment to treatment condition, and b) comparison of an income shock group to a control group with the same absolute wealth level.

6.2.1 Poverty and time preference

Numerous papers document empirically that a relationship exists between rates of time preference and poverty. In one of the earliest studies of this kind, Hausman (1979) found that poorer households had higher rates of time preference than richer households. Lawrance (1991) estimated time preferences from Euler equations in a panel of US households, and found that poorer families had significantly higher rates of time preference than richer families. Similar results were found by Harrison et al. (2002) in Denmark; Sullivan (2011) finds weak evidence in a sample from China that households with lower wealth have higher discount rates. Pender & Walker (1990) elicited rates of time preference from Indian peasants, and found that the wealth of the participants significantly predicted the rates of time preference, again with wealthier subjects being more patient. Similar results were found by Yesuf & Bluffstone (2008), who elicited time preferences from 262 households in rural Ethiopia and found that discount rates were significantly negatively correlatd with wealth variables such as the amount of land owned, the value of a household's capital stock, and, to some degree, the number of oxen owned. Note, however, that a number of studies show no significant relationship between poverty and time preferences (Cagetti, 2003; Gourinchas & Parker, 2002; Stephens & Krupka, 2006; Ogaki & Atkeson, 1997).

Nevertheless, the findings summarized above suggest that may be at least a correlational inverse relationship between rates of time preference and poverty. To test the causal role of poverty in affecting decision-making, Tanaka et al. (2010) elicited time preferences from 5340 individuals in Vietnam and fit a number of discounting models to the data, including the quasi-hyperbolic model we use here. They find that income is negatively related to delta (denoted as r in their paper), i.e., richer households show less exponential discounting; interestingly and in contrast to our findings, no such relationship is evident for the beta parameter. A particularly elegant feature of this study is the use of rainfall as an instrumental variable for income: the negative relationship between delta (but not beta) and income persists when income is instrumented using the exogenous rainfall data, and thereby establishes a causal channel from low income to high discounting. A similar approach was taken by di Falco et al. (2011), who found in a sample of 1237 households in rural Ethiopia that income shocks caused by severe droughts led to increases in the rate of time preference. Note, however, that it is not possible in this setting to disentangle the income effect of the shock from any psychological effects of the shock itself; i.e., increased rates of time preference after a shock may be entirely rational responses to the changed economic circumstances, rather than a genuine change in preferences.

In a study closely related to ours, Spears (2010) randomly assigned poor participants in India to one of four conditions: they could either be rich or poor, in the sense that they received either two (rich) or one (poor) good from a choice set of three options. In addition, each participant could either be in a "choice" or a "no choice" condition, where the former meant that participants could choose which item(s) from the choice set they wanted to receive, whereas in the latter case the items were randomly assigned. Spears then asked participants to perform two tasks that are frequently used to measure cognitive control: squeezing two handlebars for as long as possible, and performing a Stroop-like task. In this task, participants have to name the number of items in a display, which, confusingly, are numbers themselves; thus, a display might be "3 3", in which case the correct answer would be "two" since there are two items in the display. Spears found that participants performed worse on the two cognitive-control tasks if they had been randomly assigned to both the poor and the choice conditions. Thus, requiring people to make a choice appeared to deplete cognitive control, but this was only true for poor participants. Since cognitive control has frequently been related to hyperbolic discounting in the psychology literature (Shamosh et al., 2008; Shamosh & Gray, 2008), the study by Spears (2010) is similar in spirit to ours; however, note that while Spears focuses on low absolute levels of income, we hold levels constant and focus instead on negative income shocks. In addition, we measure economic choice rather than performance in psychological games.

6.2.2 Emotion and time preference

Our paper is also related to the literature that studies the effect of emotions on time preference: if negative income shocks affect the participants' affect, any effects on time preferences may be mediated by this channel. Indeed, emotions can have strong influences on time preference; Loewenstein (1996, 2000) points out that in the presence of visceral factors such as rage, people sometimes exhibit extreme discounting of future events, e.g. when politicians put their careers at risk by engaging in extramarital sex. Laboratory experiments that randomly assigned participants to particular emotion induction conditions confirm this hypothesis. For instance, Raeva et al. (2010) studied the effect of experienced regret on time preference. They elicited regret in participants by first letting them choose one of two lotteries, and then revealing that the other choice would have produced a better outcome. Control participants only saw the result of their chosen lottery, not that of the alternative, and thus could not experience regret. After this manipulation, participants completed a time preference task; those participants who had experienced regret had a lower indifference point for an outcome that was

available tomorrow, i.e. were less patient than control subjects. Conversely, participants who had experienced rejoicing, i.e. their chosen lottery was superior to the alternative, were more patient than controls. A similar paper by Ifcher & Zarghamee (AER, forthcoming) showed that positive affect, induced by a video clip of a stand-up comedian, made participants more patient than a control clip showing nature scenes.

6.2.3 Reference points and time preference

Finally, this paper is also related to the literature on reference-dependent utility and prior outcomes. Existing evidence suggests that people evaluate options against existing reference points (Köszegi & Rabin, 2006, 2007); in our experiment, it is likely that unexpected negative income shocks in our experiment put participants below the reference point, i.e. in a loss frame, while unexpected positive income shocks put them above the reference point, i.e, in a gain frame. In prospect theory, making decisions in the loss domain is associated with risk-seeking behavior, while the gain domain is associated with risk aversion (Kahneman & Tversky, 1979; Payne et al., 1980; Hershey & Schoemaker, 1980; Hershey et al., 1982; Slovic et al., 1982). Together with the theoretical and empirical relationship between risk and time preferences (Epper et al., 2011), according to which risk aversion correlates positively with present-focused time preferences (Leigh, 1986; Rachlin et al., 1991; Anderhub et al., 2001; Myerson et al., 2003; Eckel et al., 2004; Andersen et al., 2008), this literature suggests that negative income shocks might make subjects risk-seeking and therefore lead to an increase in preferences for delayed outcomes.

However, a competing account is the effect of prior losses on subsequent risky choice described by Thaler & Johnson (1990). These authors suggest that participants may "edit" the options available to them before making a choice; according to the hedonic editing rule, they do this in such a way as to make the resulting prospects appear most pleasant. In particular, this rule dictates that gains are segregated (i.e. considered independently from prior outcomes), losses are integrated with prior outcomes. Interestingly, the implication of this rule is this is risk aversion after previous losses. Thaler & Johnson present empirical evidence in favor of this view: prior monetary losses lead to increased risk aversion among their participants. In the context of the present paper, this finding suggests that negative income shocks may in fact lead to more present-focused time preferences.

In sum, it remains open to what extent poverty and negative income shocks affect time preference. We test here whether positive or negative income shocks, or absolute levels of wealth, affect time preference. To this

end, subjects receive different starting endowments which create "rich" and "poor" groups; in addition, all participants then perform a real effort task to earn money, following which subgroups of participants receive positive and negative income shocks. Subsequently we measure time preferences. We find that negative income shocks, but not positive income shocks, lead to an increase in present bias during intertemporal choice. In contrast, positive income shocks have no such effect; similarly, time preferences are not affected by mean levels of wealth ("rich" vs. "poor"). The effect of negative income shocks on time preference is specific to present bias and does not affect impatience per se. Together, these findings suggest that negative income shocks may exacerbate behavioral biases in intertemporal choice.

6.3 Methods

6.3.1 Participants

We recruited 148 healthy male participants form the subject pool of the University of Zürich. Their mean age was 22 $\pm$ 2.47 years (mean $\pm$ S.D.). We excluded students of economics and psychology, and those who were acutely or chronically ill, took medications, drugs, smoked more than 5 cigarettes a day, regularly consumed more than 60g of alcohol per day, suffered from allergies or psychiatric disorders, were in psychological or psychiatric treatment at the time of the study, or had a body mass index smaller than 18 or greater than 25. Participants were instructed to not consume medications, alcohol, or coffee, and not to engage in sexual intercourse, for 24h before the experiment. In addition, they were asked to get up at least 3h before the beginning of the experiment, and to not drink coffee, eat, smoke, or perform strenuous physical activity in the last 2h before the experiment. All participants were tested in the afternoon between 2pm and 8pm. They gave written informed consent and were reimbursed for their participation. An experimental session lasted 2h.

6.3.2 Selection of subjects

We restricted our experiment to men since controlling for ovarian cycle in women is logistically difficult. Furthermore, psychology and economics students, self-reported heavy smokers (consumption of > 5 cigarettes per day), heavy alcohol consumers (consumption of > 60 g alcohol per day) and drug users were excluded. Participants were German native speakers and would stay in Zurich at least for the next 12 months (for payment of their reward).

The study was approved by the ethical committee of the University of Zurich and all participants provided written informed consent. Participants received a variable reimbursement for their participation, depending on the choices they made during the experiment.

6.3.3 Procedure

At the beginning of the experiment, each participant was informed about the nature of the tasks to be performed and trained to use the saliva sampling devices (Salivettes). Participants were also told that they might experience a change in their wealth during the real effort task that they would perform. They were told that they would experience either exactly zero or exactly one such wealth change, but were not told the timing, magnitude, or direction of this change.

After these instructions, each participant completed a PANAS questionnaire (Watson et al., 1988) and five visual analog scales, which asked to what extent participants currently felt a) stressed, b) self-control, c) optimistic, d) self-confident, e) government vs. individual responsibility. Participants marked their current feelings on a 10 cm line; responses were coded as between 0 and 100.

Each participant was randomly assigned to one of four treatment conditions, unbeknownst to them: "stay rich"; "stay poor"; "negative income shock"; "positive income shock". When the experiment began, participants in the "stay rich" and "negative income shock" groups had a high initial endowment of 1000 points; in contrast, the "stay poor" and "positive income shock" groups had a low initial endowment of 100 points. 70 points were converted into 1 CHF at the end of the experiment and paid out.

Throughout the experiment, participants were informed of their own current wealth through bars and numbers on the screen; the size of the bar corresponded to the current wealth of the participant. In addition, bars were also shown for current maximum wealth, minimum wealth, and average wealth across all participants. Thus, participants could continually keep track of their own wealth, and its relation to the wealth of the entire group of participants. Bars were always normalized to the maximum wealth bar for ease of display.

6.3.4 Real effort task

Participants then participated in a real effort task for 15 periods, which resembled that used by Abeler (2009). Each period lasted 2 minutes. The task consisted of counting the number of zeros in a 7 x 5 random table to

zeros and ones, which was presented on the left side of the screen. The right side of the screen displayed the wealth variables described above – own wealth, and maximum, minimum, and average wealth of all participants; the purpose of displaying this information even during task performance was to make own wealth in comparison to that of the entire group as salient as possible. After counting the zeros in a given table, participants entered their answer in a text field at the bottom of the screen. The next table was then displayed, without feedback about performance to minimize learning effects. Participants counted as many tables as they could within each 2 minute period, and earned 5 points for every correctly counted table. After each period, the accumulated points from the period were added to the wealth of the participant and displayed for 20 seconds in the middle of the screen, again also showing minumu, maximum, and average wealth. After these 20 seconds, the next period began.

Participants played 15 periods of the real effort task, which lasted 35 minutes. After 15 periods of earning income, the two income shock groups received their income shocks. The timing, magnitude, and direction of these shocks was unanticipated; however, participants were informed at the beginning of the experiment that they might experience a sudden change in their wealth levels. No justification was given for the income shocks; participants were informed of the shock through a screen that read "Your income has decreased by x points" or "Your income has increased by y points". The participants in the "stay rich" and "stay poor" groups were also told at the beginning of the experiment that they might experience sudden changes in their wealth levels during the experiment, but they did not receive income shocks after period 15, nor were they told when the other participants experienced the income shocks.

The magnitude and direction of the income shock for the "downward income shock group" was such that the post-shock average wealth of this group was equal to the pre-shock average wealth of the "stay poor" group. Similarly, the magnitude and direction of the income shock for the "upward income shock group" was such that the post-shock average wealth of this group was equal to the pre-shock average wealth of the "stay rich" group. Put differently, the two groups switched positions from the "poor" into the "rich" group, and vice-versa. This allows us to then compare the effect of income shocks on economic choice, holding constant current wealth: comparing the behavior of the "negative income shock" group to the "stay poor" group reveals the effect of a negative income shock, holding constant current wealth, while comparing the behavior of the positive income shock group to the "stay rich" group reveals the effect of a positive income shock, again holding constant current wealth.

After receiving the income shock, participants were again presented with their updated wealth and the maximum, minimum, and average wealth across participants. This information was displayed for one minute to make their new wealth salient to participants in the shock groups. Participants then played two more periods of the real effort task; the purpose of these two periods was again to make participants fully aware of their new wealth situation and their position relative to others.

After period 17, participants performed the behavioral tasks of interest, in particular, an intertemporal choice task and a BDM auction. The following sections describe these tasks in greater detail.

6.3.5 Intertemporal Choice Task

Participants performed three blocks of an intertemporal choice task with varying delays, where decisions between a sooner smaller reward and a later larger reward were offered. In the first two blocks subjects had the choice between a smaller reward tomorrow, and a larger reward in a) 6 months and 1 day, or b) 12 months and 1 day. The short delay was set to "tomorrow" rather than "today" to keep transaction costs the same for sooner and later payments. In the last block, subjects chose between a smaller reward in 6 months and 1 day, and a larger reward in 9 months and 1 day. Each block consisted of 6 binary choice trials, resulting in a total of 18 trials. The larger reward was kept constant at an amount of 30 Swiss Francs (CHF), while the sooner smaller reward started at CHF 15 and was then adjusted with a titration method according to the choices the subject made.

Reimbursement consisted of a flat rate of CHF 10 and a variable payment depending on participants' choices. In particular, as was explained to the participants at the beginning of the study, one of all the choices made was randomly selected at the end of the study, and the chosen option on this trial was paid out for real, i.e., participants could pick up the chosen amount on the chosen day of delivery, using a voucher valid at the University cashier's office. As mentioned, transaction costs were kept constant by setting the soonest outcome to "tomorrow".

Titration is a standard method for identifying time preferences in the discounting literature (Mazur, 1988; Green & Myerson, 2004; Kable & Glimcher, 2007; Rachlin et al., 1991). The titration worked as follows: for each choice of the later reward, the sooner reward was increased by half the difference between it and 30 CHF; for instance, if a subject chose CHF 30 in 12 months and 1 day over CHF 15 tomorrow, the next trial would offer the subject a choice between CHF 30 in 12 months and 1 day and CHF 22.50 tomorrow; if the subject still chose CHF 30 in 12 months and 1 day, the next

offer would be CHF 30 in 12 months and 1 day vs. CHF 26.25 tomorrow, and so on. For each choice of the sooner reward, the sooner reward was decreased by half of the difference between it and the previously offered soon reward. For instance, if a subject chose CHF 15 tomorrow over CHF 30 in 12 months and 1 day, the next trial would offer the subject a choice between CHF 7.50 tomorrow and CHF 30 in 12 months and 1 day; if the subject chose CHF 7.50 tomorrow, the next offer would be CHF 3.75 tomorrow vs. CHF 30 in 12 months and 1 day, and so on. The titration procedure lasted for 6 trials at each combination of delays; this means that each indifference point was identified to a precision of CHF 0.23 (CHF 15 * 0.5^6, i.e. the initial difference between CHF 15 and CHF 30/ CHF 0 was halved six times). The amount of the sooner reward at the end of this titration procedure was taken as the indifference point for the particular delay combination, i.e. the amount of the sooner smaller reward where participants switched between the smaller sooner and the later larger reward.

This procedure resulted in an individual discount function for each subject, which was used as the basis for fitting parameters of several models of intertemporal choice, described below. Note that this procedure collapses subjects' choices in the time preference task into one or two parameters; thus, each subject entered the statistical analysis only once, i.e. we are not using multiple (non-independent) data points for each subject.

Possible serial correlation and order effects in subjects' responses were controlled for by randomizing the order of trials across blocks, i.e. the order in which the various indifference points were determined.

Note that the soonest option subjects could choose in the intertemporal choice task was "tomorrow". One may ask whether this delay can be considered small enough to be useful in identifying present bias. We chose this design for the following reasons:

First, we found it difficult to include an earlier reward in the design without confounding transaction costs: the chosen option on one of the trials in the intertemporal choice task was paid out for real, i.e., participants could pick up the chosen amount on the chosen day of delivery, using a voucher valid at the University cashier's office. If the smallest delay was "today", choosing this option would result in lower transaction costs compared to choosing a more delayed option, because subjects are already at the University, while at any other delay than today (i.e. "tomorrow", but also in several months) subjects may have to travel to the University specifically to pick up their payment. Therefore, in this case we would have been unable to dissociate transaction costs from present bias.

Second, we did consider other forms of payment than cash vouchers, but they all suffered from similar problems: we judged that getting a check or

cash on the day vs. receiving a check or cash in the mail later did not equate the perceived risk of the transaction; bank transfers cannot be effected on the same day and also have to be picked up at the bank before they can be consumed; Amazon vouchers cannot be turned into consumption immediately because of the delays associated with mail orders; mobile phone money transfers and pre-paid debit cards are not available in Switzerland. Thus, the "tomorrow" option seemed to us the cleanest way of eliciting time preference without risk of transaction cost confounds.

Third, note that even with a "today" option, when using money as a reward, it is almost impossible to study true present bias in the sense of the immediate present; at best, one could hope for a time frame of a few hours, since subjects are unable to spend the money they earn in the experiment until they leave the lab. Thus, even a "today" option would not fully address the disconnect between the "present" and the earliest time at which the reward can actually be consumed.

Fourth, to the extent that we observe present bias in our behavioral data (we consistently find beta < 1), we argue that people actually do consider tomorrow as part of the extended present. Thus, in our view it is likely that, even if it were possible to overcome the difference in transaction costs, similar results would be found if all the payoffs had been one day earlier (i.e. today, in 3 months, in 6 months etc.).

Fifth, note that the delays in our temporal choice task are relatively long, ranging from 6 months to 12 months. In comparison, the delay between today and tomorrow is very small in magnitude, making it likely that tomorrow is in fact interpreted as part of the extended present by our subjects. In support of this claim, comparable studies where the soonest payoff was on the same day find similar discount rates as we do when the soonest payoff is tomorrow (Benizon et al., 1989; Thaler, 1981; Frederick et al., 2002). In particular, estimates of the beta parameter reported in the literature involving, among others, immediate outcomes in real-life or laboratory situations are very similar to those obtained for our control group (Laibson et al., 1998, 2007).

Finally, note that if "tomorrow" is not considered part of the extended present by our subjects, it is likely that we in fact underestimated the behavioral effect of negative income shocks on present bias. Conversely, we would predict that the effect of negative income shocks on present bias would be stronger if a "today" option was included.

6.3.6 BDM Auction Task

We next asked whether the income shocks changed participants' reservation wage; for instance, might participants who just lost a substantial proportion of their wealth be more willing to work at a lower wage? We therefore conducted a Becker-deGroot-Marschak (1964) auction in which participants could bid against the computer on the opportunity to complete the real effort task for another eight periods. Participants entered their bid into a text field, the computer played the auction immediately, and winning participants performed the real effort task for another 8 periods, while the remainder of the subjects waited until they had completed the experiment. The advantage of this type of auction is that it is incentive-compatible and elicits subjects' true willingness to pay for playing a further eight periods.

The computer bid was randomly drawn from a uniform distribution between 0 and the expected earnings from a further 8 periods of play, based on performance of each subject in the first 15 periods of the real effort task. If participants' willingness exceeded the computer bid, they could perform the real effort task for the remaining 8 periods.

At the end of the study, participants completed another PANAS questionnaire and five visual-analog scales (see above). Finally, they completed a socioeconomic questionnaire and the Barratt Impulsiveness Scale (Patton et al., 1995), and were paid and excused.

6.3.7 Model Fits

For every subject and every delay level, we determined the amount at which a subject was indifferent between the immediate and delayed option (in items with an immediate option), or the delayed and the very delayed option (in items with an added front-end delay) based on the individual switching points (see above). This allowed us to express the subjective value of the delayed reward as a fraction of the subjective value of the immediate reward. We then plotted the relative values of the delayed rewards as a function of time. Next, for every subject, we fitted three different models to the obtained indifference points.

Beta-delta quasi-hyperbolic discount model. Laibson's beta-delta model (Laibson, 1997) was fitted to the indifference points to obtain an estimate of the degree of impatience and present bias:

$$V_t = v\left(r_t\right) + \sum_{\tau=1}^{T-t} \delta_t v\left(r_{t+\tau}\right),$$

where V_t indicates the discounted value at time t of a stream of rewards r with subjective values v as a function of time τ. This equation contains a constant, exponential discount function whose discount rate is $\ln \frac{1}{\delta}$, thus whose steepness can be characterized by δ. The β parameter deflects the exponential discount curve and its inverse can be interpreted as the extra weight added to immediate rewards. Hence, δ can be interpreted as measuring impatience and β as measuring present bias.

6.3.8 Statistical Analyses

The effect of negative income shocks on the outcome variables was assessed using OLS regressions of the following form:

$$y_i = \beta_0 + \beta_1 negshock_i + \beta_2 \mathbf{X}_i + u_i$$
$$y_i = \beta_0 + \beta_1 posshock_i + \beta_2 \mathbf{X}_i + u_i,$$

where y_i are the three outcome variables, i.e. β_i, δ_i, and bdm_i. β_i and δ_i are the present bias and impatience parameters, respectively, in Laibson's discounting model, $negshock_i$ and $posshock_i$ are dummy variables indicating whether subject i was in the "negative income shock" group, the positive income shock group, or the control group, $\mathbf{X}_i$ is a vector of covariates including age, body mass index, and number of siblings, and u_i is the error term. The regression with the negshocki term was only run for subjects in the "stay poor" and "negative income shock" groups, since these two groups are identical in terms of their average wealth at the beginning of the economic choice tasks. Similarly, the regression with the posshocki term was only run for subjects in the "stay rich" and "positive income shock" groups.

6.4 Results

The main question of this study was whether income shocks affect time preference, while wealth levels are held constant. Our design allows us to test this hypothesis as follows: first, comparing the "negative income shock" group to the "stay poor" group after the income shock identifies the effect of negative income shocks on time preference; second, comparing the "positive income shock" group to the "stay rich" group after the income shock identifies the effect of positive income shocks. Crucially, the two groups being compared have identical wealth levels after the income shock, thus enabling us to compare the effect of income shocks on preferences without confounds from different wealth levels.

To ascertain that the income shock manipulations worked as intended, we first report the evolution of wealth levels while performing the real effort task. The "stay rich" and "negative income shock" groups started the experiment with an endowment of 1000 points; during the first 15 periods, the average wealth level in these two groups grew to 1948.38 ± 28.60 (mean ± SEM) points, with no significant difference between groups (as is expected, since the groups were identical up to that point; data not shown). Similarly, the "stay poor" and "positive income shock" groups started the experiment with an endowment of 100 points; during the first 15 periods, the average wealth level in these two groups grew to 1029.46 ± 27.17 points, again with no significant difference between the groups. The magnitude and direction of the income shock was -918.92 ± 5.84 for the "negative income shock" group, and +918.92 ± 5.84 for the "positive income shock" group. Note that these shocks are equal in magnitude and opposite in sign by design, since the two groups simply switched positions; i.e., each participant in the "negative income shock" group lost the same number of points, and each participant in the "positive income shock" group gained the same number of points. The non-zero variance of the income shocks stems from the fact that the pre-shock difference between the groups differed somewhat across experimental sessions. In sum, the real effort task and the experimental manipulation of wealth levels through income shocks worked as intended. Figure 6.4.1 shows the evolution of wealth levels as a function of period throughout the experiment; it can be seen that the post-shock wealth levels match exactly those of the "stay rich" and "stay poor" groups, respectively.

We next asked whether the negative income shock indeed had an effect on time preferences. To this end, we fit participants' responses in the time preference task using Laibson's (1997) quasi-hyperbolic discounting model. The results are shown in Figure 6.4.1 and Table 6.4.1. It can be seen that participants in the "negative income shock" group exhibit lower post-shock levels of beta than participants in the "stay poor" group; specifically, the mean beta parameter in the "stay poor" control group is 0.77 ± 0.03, while that in the "negative income shock" group is 0.65 ± 0.04. Crucially, the wealth levels of these two groups are identical at the time of testing, and thus any differences in beta must be attributed to the negative income shock per se, rather than to wealth differences. Table 6.4.1 shows the results of an OLS regression effect of Laibson's present bias parameter beta on negative income shocks, for only the participants in the "negative income shock" and "stay poor" groups. Each of the three regression specifications reports different sets of control variables. In all cases, the coefficient on beta is significant and negative, suggesting that negative income shocks decrease beta. The effect is also economically significant; the magnitude of the coefficients is around

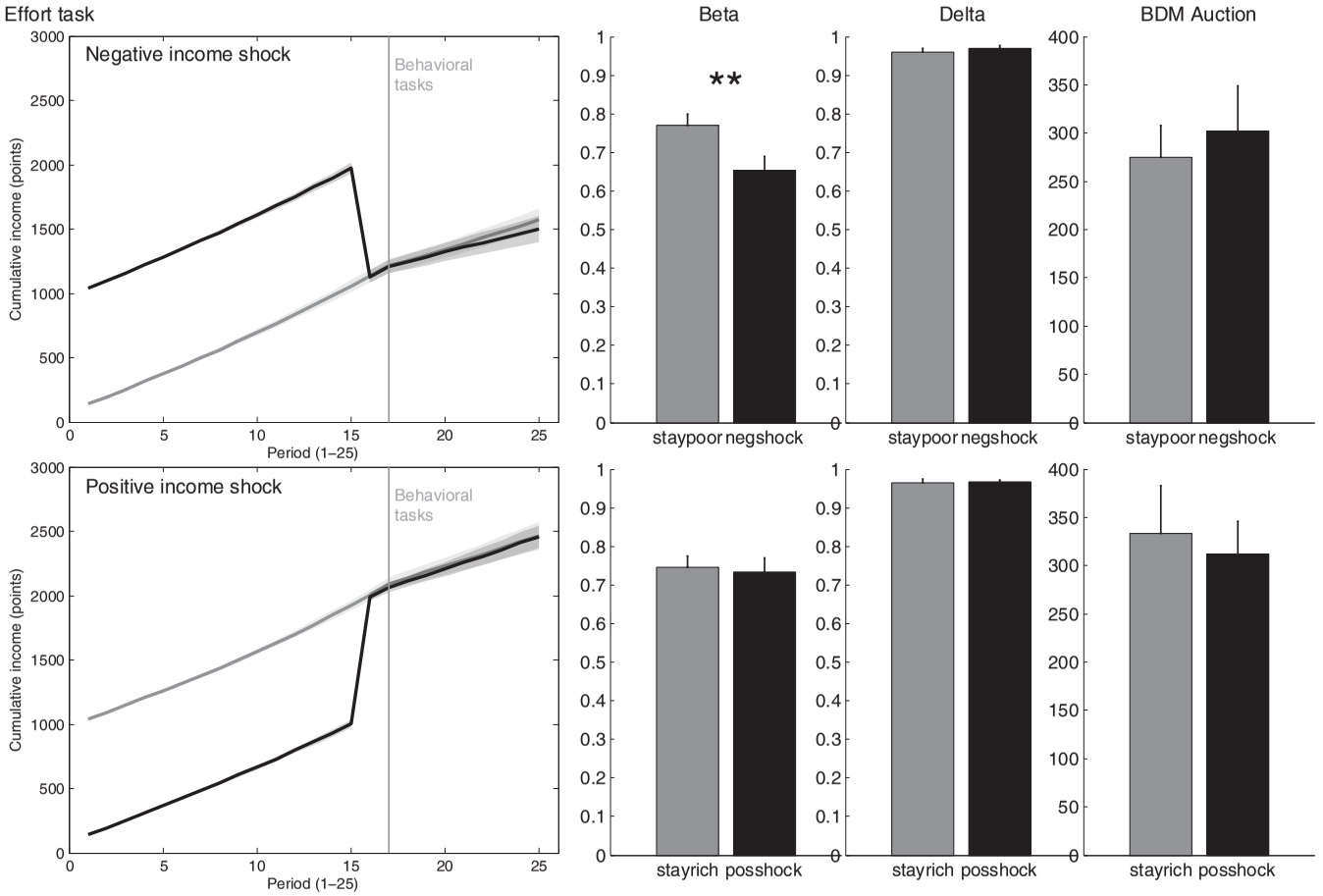

Figure 6.4.1: Effect of income shocks on time preference and reservation wage. Left panels: cumulative income during the real effort task. The solid lines show the mean cumulative income across periods for each group; the shaded areas indicate 1 SEM. Remaining panels: effect of negative (top row) and positive (bottom row) income shocks on the present bias parameter beta, the impatience parameter delta, and reservation wage as measured by the BDM auction bid.

VARIABLES	(1) model1 beta	(2) model2 beta	(3) model3 beta
negshock	-0.116**	-0.116**	-0.106**
	(0.0490)	(0.0492)	(0.0524)
income		-5.77e-06	3.61e-05
		(5.37e-05)	(7.30e-05)
age			-0.0152
			(0.0107)
bmi			-0.000536
			(0.0132)
numsibs			-0.00772
			(0.0192)
Constant	0.769***	0.773***	1.095***
	(0.0307)	(0.0484)	(0.325)
Observations	74	74	74
R-squared	0.073	0.073	0.100

Table 6.4.1: Effect of Negative Income Shocks on Beta. OLS regression of the present bias parameter beta on having received a negative income shock, for the "stay poor" and "negative income shock" conditions only. These participants had the same wealth levels when they performed the time preference task; thus any differences in behavior are attributable to the negative income shock. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

0.1 in all specficiations, corresponding to a change in beta induced by the negative income shock of about $0.1/0.77 = 13\%$.

Is this effect of income shocks on beta specific to negative income shocks, or does it also obtain for positive income shocks? Figure 6.4.1 and Table 6.4.2 show the effect of positive income shocks on beta: in the "positive income shock" group, mean beta is 0.73 ± 0.04, while in the "stay rich" control group, it is 0.75 ± 0.03. Table 6.4.2 shows that this difference is not statistically significant.

We next tested whether the effect of negative income shocks was specific to the present bias parameter beta, or whether it also extended to the impatience parameter delta. We find no effect of income shocks on the impatience parameter delta: in the "negative income shock" group, mean delta is 0.97 $\pm$ 0.01, while in the "stay poor" group, it is 0.96 $\pm$ 0.01. Table 6.4.3 shows

VARIABLES	(1) model1 beta	(2) model2 beta	(3) model3 beta
posshock	-0.0109	-0.00685	-0.00982
	(0.0459)	(0.0454)	(0.0457)
income		4.76e-05	5.65e-05
		(3.71e-05)	(4.34e-05)
age			-0.0125
			(0.00954)
bmi			0.00907
			(0.0113)
numsibs			-0.00325
			(0.0203)
Constant	0.745***	0.709***	0.788**
	(0.0292)	(0.0383)	(0.370)
Observations	74	74	74
R-squared	0.001	0.016	0.049

Table 6.4.2: Effect of Positive Income Shocks on Beta. OLS regression of the present bias parameter beta on having received a positive income shock, for the "stay rich" and "positive income shock" conditions only. These participants had the same wealth levels when they performed the time preference task; thus any differences in behavior are attributable to the positive income shock. Robust standard errors in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

VARIABLES	(1) model1 delta	(2) model2 delta	(3) model3 delta
negshock	0.00922	0.00929	0.00893
	(0.0109)	(0.0109)	(0.0120)
income		4.29e-06	-3.93e-06
		(1.31e-05)	(1.20e-05)
age			0.00369*
			(0.00192)
bmi			-0.00303
			(0.00246)
numsibs			0.00129
			(0.00545)
Constant	0.961***	0.958***	0.949***
	(0.00820)	(0.0116)	(0.0660)
Observations	74	74	74
R-squared	0.010	0.011	0.046

Table 6.4.3: Effect of Negative Income Shocks on Delta. OLS regression of the impatience parameter delta on having received a negative income shock, for the "stay poor" and "negative income shock" conditions only. These participants had the same wealth levels when they performed the time preference task; thus any differences in behavior are attributable to the negative income shock. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

that this difference is not significant. Similarly, positive income shocks have no effect on delta: in the "positive income shock" group, mean delta is 0.97 ± 0.01, while in the "stay rich" group, it is 0.97 ± 0.01. Again this difference is not statistically significant, as shown in Table 6.4.4.

Thus, we find two dissociations in the effect of income shocks affect on time preference: first, only negative income shocks affect time preference, but positive income shocks do not; second, income shocks affect only present bias, but not impatience.

We next asked whether income shocks also affect reservation wages. To this end, participants were given the opportunity to make a bid in a BDM auction on the right to play the real effort task for a further 8 periods after the end of the behavioral tasks that followed period 17. The bid made by participants is an incentive-compatible estimate of the true value to each par-

VARIABLES	(1) model1 delta	(2) model2 delta	(3) model3 delta
posshock	0.000892	-0.00104	-0.00364
	(0.0112)	(0.0107)	(0.0102)
income		-2.27e-05***	-2.20e-05**
		(8.30e-06)	(9.80e-06)
age			-0.000357
			(0.00265)
bmi			-0.00102
			(0.00248)
numsibs			0.00884
			(0.00556)
Constant	0.965***	0.982***	0.999***
	(0.00956)	(0.00941)	(0.0963)
Observations	74	74	74
R-squared	0.000	0.059	0.095

Table 6.4.4: Effect of Positive Income Shocks on Delta. OLS regression of the impatience parameter delta on having received a positive income shock, for the "stay rich" and "positive income shock" conditions only. These participants had the same wealth levels when they performed the time preference task; thus any differences in behavior are attributable to the positive income shock. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

VARIABLES	(1) model1 bdm_auction	(2) model2 bdm_auction	(3) model3 bdm_auction
negshock	27.08	26.48	29.49
	(57.42)	(57.37)	(59.87)
income		-0.0358	0.0626
		(0.0582)	(0.0600)
age			-19.83*
			(9.947)
bmi			-8.596
			(14.54)
numsibs			-47.24***
			(17.36)
Constant	274.6***	298.4***	937.2***
	(33.20)	(49.59)	(303.5)
Observations	74	74	74
R-squared	0.003	0.007	0.094

Table 6.4.5: Effect of Negative Income Shocks on BDM Auction Offer. OLS regression of the BDM auction offer on having received a negative income shock, for the "stay poor" and "negative income shock" conditions only. These participants had the same wealth levels when they performed the BDM auction; thus any differences in behavior are attributable to the negative income shock. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

ticipant of playing a further 8 periods, and is thus a proxy for the reservation wage of our participants. We found that income shocks did not affect BDM bids: participants in the "negative income shock" group made average bids of 301.73 ± 46.85, while those in the "stay poor" group made average bids of 274.65 ± 33.20. Similarly, participants in the "positive income shock" group made average offers of 311.59 ± 34.50, while those in the "stay rich group" made offers of 333.68 ± 49.03. None of these differences were statistically significant, as shown in Table 6.4.5 for negative income shocks and Table 6.4.6 for positive income shocks. Thus, income shocks had no statistically significant effect on reservation wages in our study, as measured by BDM bids.

Finally, we asked whether the effect of negative income shocks on the present bias parameter beta might be mediated through effects of the negative

	(1)	(2)	(3)
	model1	model2	model3
VARIABLES	bdm_auction	bdm_auction	bdm_auction
posshock	-22.08	-26.06	-22.81
	(59.94)	(60.50)	(64.36)
income		-0.0468	-0.0517
		(0.0484)	(0.0520)
age			3.217
			(13.27)
bmi			4.961
			(15.17)
numsibs			-13.55
			(32.92)
Constant	333.7***	368.9***	212.1
	(49.03)	(64.84)	(382.4)
Observations	74	74	74
R-squared	0.002	0.011	0.015

Table 6.4.6: Effect of Positive Income Shocks on BDM Auction Offer. OLS regression of the BDM auction offer on having received a positive income shock, for the "stay rich" and "positive income shock" conditions only. These participants had the same wealth levels when they performed the BDM auction; thus any differences in behavior are attributable to the positive income shock. Robust standard errors in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

income shock on psychological outcomes. We therefore computed the after-before difference of participants' responses on the five visual analog scale questions, and the after-before differences in positive and negative affect as measured by the PANAS scale. The VAS questions elicited self-reported stress, self-control, optimism, self-esteem, and responsibility. The results of OLS regressions of these variables on negative income shocks are shown in Table 6.4.7; we observe a mild negative effect of negative income shocks on self-reported stress, but no other significant effects. Similarly, the results of OLS regression of the psychological outcomes on positive income shocks are shown in Table 6.4.8; we observe a significant negative effect of positive income shocks on self-reported stress levels, but no other significant effects. Thus, participants in the income shock groups appear to report lower levels of stress than those in the control groups; we discuss this finding further below.

6.5 Discussion

The purpose of this study was to test whether income shocks affect preferences. It has already been shown that a correlation exists between income levels and time preference; in particular, poor people tend to be more impatient than rich people (Lawrance,1991; Harrison et al., 2002; Sullivan, 2011; Pender & Walker, 1990; Yesuf & Bluffstone, 2008; but see Cagetti, 2003; Gourinchas & Parker, 2002; Stephens & Krupka, 2006; Ogaki & Atkeson, 1997). These studies suffer from the familiar correlation-causality problem: it remains unclear whether poverty actually causes changes in time preferences; in addition, it is not clear to what extent observed differences in discounting behavior actually reflect differences in preferences, or whether they may instead reflect actual or perceived environmental constraints in conditions of poverty. The former question has been addressed to some extent by studies using rainfall data as a source of exogenous variation in income, allowing identification of a causal effect from wealth to discounting behavior (Tanaka et al., 2010; di Falco et al., 2011). However, the second problem persists: it remains unclear whether these observed differences in behavior are reflective of preferences or beliefs.

To address this question, we conducted a laboratory experiment in which subjects receive either positive or negative income shocks; crucially, after the shock they have the same level of wealth as a control group that did not receive a shock, allowing a comparison of time preferences across groups which differ only in whether or not they received a shock, not in their levels of wealth. We find that negative income shocks, but not positive income

VARIABLES	(1) model1 stress	(2) model2 selfcontrol	(3) model3 Optimism	(4) model4 selfesteem	(5) model5 responsibility	(6) model6 panas_pos	(7) model7 panas_neg
negshock	-9.703*	3.922	-1.796	3.639	1.398	-0.250	0.0862
	(5.784)	(4.131)	(4.461)	(3.590)	(2.831)	(0.167)	(0.104)
Constant	18.09***	-5.657*	-6.057*	-1.257	-1.457	-0.0658	0.192**
	(4.197)	(2.888)	(3.376)	(2.889)	(2.190)	(0.131)	(0.0793)
Observations	69	69	69	69	69	74	74
R-squared	0.040	0.013	0.002	0.015	0.004	0.030	0.009

Table 6.4.7: Effect of Negative Income Shocks on Psychological Outcomes. OLS regression of psychological outcomes on having received a negative income shock, for the "stay poor" and "negative income shock" conditions only. Each dependent variable is the difference between participants' answers on the psychological scales after the income shock vs. before the beginning of the real effort task. Specifically, the dependent variable in the first five columns is the after vs. before difference in self-reported stress, self-control, optimism, self-esteem, and responsibility, respectively; in the last two columns, it is the after vs. before difference in positive affect (column 6) and negative affect (column 7). Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

VARIABLES	(1) model1 stress	(2) model2 selfcontrol	(3) model3 optimism	(4) model4 selfesteem	(5) model5 responsibility	(6) model6 panas_pos	(7) model7 panas_neg
posshock	-11.17**	0.189	4.173	5.344	-2.087	0.00661	-0.110
	(5.205)	(3.475)	(4.320)	(3.767)	(1.773)	(0.164)	(0.0704)
Constant	11.26***	0.114	-1.143	-1.829	0.0571	-0.126	0.118**
	(3.497)	(2.654)	(3.117)	(2.380)	(1.385)	(0.113)	(0.0541)
Observations	68	68	68	68	68	74	74
R-squared	0.065	0.000	0.014	0.030	0.020	0.000	0.033

Table 6.4.8: Effect of Positive Income Shocks on Psychological Outcomes. OLS regression of psychological outcomes on having received a positive income shock, for the "stay rich" and "positive income shock" conditions only. Each dependent variable is the difference between participants' answers on the psychological scales after the income shock vs. before the beginning of the real effort task. Specifically, the dependent variable in the first five columns is the after vs. before difference in self-reported stress, self-control, optimism, self-esteem, and responsibility, respectively; in the last two columns, it is the after vs. before difference in positive affect (column 6) and negative affect (column 7). Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

shocks, affect time preferences; in addition, what is affected is the present bias parameter beta, but not the impatience parameter delta. Furthermore, we find no effect of income shocks on reservation wages in a BDM auction, nor on psychological outcomes such as positive and negative affect, perceived self-control, optimism, self-esteem, and responsibility.

However, we do find a mild effect of income shocks on self-reported stress. In particular, we observe a weakly significant negative effect of negative income shocks on stress, i.e. participants in the "negative income shock" group showed somewhat lower self-reported levels of stress after than before the shock in comparison to the control group. This finding is somewhat surprising; it is possible that it may reflect a coping strategy to deal with the stressful experience of having received a substantial negative income shock. Less surprising is the highly significant negative effect of positive income shocks on self-reported stress; participants who received positive income shocks report lower levels of stress after the income shock compared to before the income shock and compared to the control group. This finding is plausible in light of the fact that subjects perceived the positive income shocks as a pleasant event; however, it should be noted that this psychological effect apparently was not strong enough to also manifest itself in altered economic preferences, as we observe no detectable effect of positive income shocks on time preferences.

Our findings also distinguish between to alternative accounts of the effect of reference points on time and risk preferences (Köszegi & Rabin, 2006, 2007). The first of these is that the negative income shock put participants below their reference point, which was built up during the real effort task. It has been argued that being below the reference point induces risk-seeking behavior (Kahneman & Tversky, 1979); this would predict a decrease in present-focused time preference because temporally remote outcomes are riskier. Another account by Thaler & Johnson (1990), in contrast, suggests that prior losses introduce risk-averse behavior; this effect in turn should induce present-focused time preferences. Our study therefore contributes to the literature on the effect of reference points on preferences by showing that the second of these accounts is more plausible.

Together, our findings suggest that negative income shocks have a direct effect on economic preferences; in particular, they increase present bias in intertemporal choice. It is widely held that humans are more present-biased than is good for their own long-run welfare; the mechanism we present here suggests a positive feedback loop that may account for some of this effect. In particular, if falling into poverty leads to present bias in intertemporal choice, then this effect is likely to perpetuate poverty by leading to imprudent intertemporal decisions.

Bibliography

[1] Anderhub, V., Güth, W., Gneezy, U., & Sonsino, D. (2001). On the Interaction of Risk and Time Preferences: An Experimental Study. German Economic Review, 2(3), 239-253. doi:10.1111/1468-0475.00036

[2] Andersen, S., Harrison, G. W., Lau, M. I., & Rutström, E. E. (n.d.). Eliciting Risk and Time Preferences. SSRN eLibrary. Retrieved from http://papers.ssrn.com/sol3/papers.cfm?abstract_id=872325

[3] Banerjee, A. V. (2001). Contracting Constraints, Credit Markets and Economic Development. SSRN eLibrary. Retrieved from http://papers.ssrn.com/sol3/papers.cfm?abstract_id=306990

[4] Bardhan, P., 1996. Research on Poverty and Development Twenty Years after Redistribution with Growth. Proceeding of the Annual World Bank Conference on Development Economics 1995. Supplement to the World Bank Economic Review and the World Bank Research Observer, pp. 59–72.

[5] Becker, G. M., DeGroot, M. H., & Marschak, J. (1964). Measuring utility by a single-response sequential method. Behavioral Science, 9(3), 226-232.

[6] Becker, G. S., & Mulligan, C. B. (1997). The Endogenous Determination of Time Preference. The Quarterly Journal of Economics, 112(3), 729 -758. doi:10.1162/003355397555334

[7] Benzion, U., Rapoport, A., & Yagil, J. (1989). Discount Rates Inferred from Decisions: An Experimental Study. Management Science, 35(3), 270 -284. doi:10.1287/mnsc.35.3.270

[8] Bertrand, M., Mullainathan, S., & Shafir, E. (2004). A behavioral-economics view of poverty. American Economic Review: Papers and Proceedings, 94(2), 419-423.

[9] Cagetti, M. (2003). Wealth Accumulation Over the Life Cycle and Precautionary Savings. Journal of Business and Economic Statistics, 21(3), 339-353. doi:10.1198/073500103288619007

[10] Chakrabarty, D. (2000). Poverty Traps and Growth in a Model of Endogenous Time Preference. SSRN eLibrary. Retrieved from http://papers.ssrn.com/sol3/papers.cfm?abstract_id=248476

[11] Dasgupta, P. (1997). Environmental and resource economics in the world of the poor. Resources for the Future.

[12] Di Falco, S., Damon,, M., & Kohlin, G. (2011). Environmental Shocks, Rates of Time Preference and Conservation: A Behavioural Dimension of Poverty Traps? Working paper.

[13] Drichoutis, A., & Nayga, R. (2010). Eliciting risk and time preferences under induced mood states. Retrieved from http://mpra.ub.uni-muenchen.de/28338/

[14] Eckel, C., Johnson, C., & Montmarquette, C. (2004). Saving decisions of the working poor: Short- and long-term horizons. In Research in experimental economics, 10, 219–260.

[15] Epper, T., Fehr-Duda, H., & Bruhin, A. (2011). Viewing the future through a warped lens: Why uncertainty generates hyperbolic discounting. Journal of Risk and Uncertainty, 43(3), 169-203. doi:10.1007/s11166-011-9129-x

[16] Frederick, S., Loewenstein, G., & O'donoghue, T. (2002). Time Discounting and Time Preference: A Critical Review. Journal of Economic Literature, 40(2), 351-401. doi:10.1257/002205102320161311

[17] Gourinchas, P., & Parker, J. A. (2002). Consumption Over the Life Cycle. Econometrica, 70(1), 47-89. doi:10.1111/1468-0262.00269

[18] Green, L., & Myerson, J. (2004). A discounting framework for choice with delayed and probabilistic rewards. Psychological Bulletin, 130(5), 769-792. doi:10.1037/0033-2909.130.5.769

[19] Gunnar Kohlin, Salvatore Di Falco, & Luca Di Corato. (2011). Environmental Shocks and Rates of Time Preference: A Behavioural Dimension of Poverty Traps? Gothenburg University. Retrieved from http://www.webmeets.com/EAERE/2011/prog/viewpaper.asp?pid=914

[20] Hall, Crystal. 2008. \Decisions under Poverty: A Behavioral Perspective on the Decision Making of the Poor." Ph.D. dissertation, Princeton University.

[21] Harris, C., & Laibson, D. (2001). Dynamic Choices of Hyperbolic Consumers. Econometrica, 69(4), 935-957.

[22] Harrison, G. W., Lau, M. I., Rutstrom, E. E., & Williams, M. B. (2002). Estimating Individual Discount Rates in Denmark: A Field Experiment (Artefactual Field Experiments). The Field Experiments Website. Retrieved from http://ideas.repec.org/p/feb/artefa/0053.html

[23] Hausman, J. A. (1979). Individual Discount Rates and the Purchase and Utilization of Energy-Using Durables. The Bell Journal of Economics, 10(1), 33-54. doi:10.2307/3003318

[24] Hershey, J. C., & Schoemaker, P. J. H. (1980). Risk Taking and Problem Context in the Domain of Losses: An Expected Utility Analysis. The Journal of Risk and Insurance, 47(1), 111-132. doi:10.2307/252685

[25] Hershey, J. C., Kunreuther, H. C., & Schoemaker, P. J. H. (1982). Sources of Bias in Assessment Procedures for Utility Functions. Management Science, 28(8), 936-954.

[26] Hill, M. N., McLaughlin, R. J., Pan, B., Fitzgerald, M. L., Roberts, C. J., Lee, T. T.-Y., Karatsoreos, I. N., et al. (2011). Recruitment of prefrontal cortical endocannabinoid signaling by glucocorticoids contributes to termination of the stress response. The Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 31(29), 10506-10515. doi:10.1523/JNEUROSCI.0496-11.2011

[27] Holden, S.T., Shiferaw, B., & Wik, M. (1998). Poverty, credit constraints, and time preferences: of relevance for environmental policy? Environment and Development Economics, 3, 105-130.

[28] Ifcher, J., & Zarghamee, H. (2008). Happiness and Time Preference: The Effect of Positive Affect in a Random-Assignment Experiment. SSRN eLibrary. Retrieved from http://papers.ssrn.com/sol3/papers.cfm?abstract_id=1491247

[29] Joëls, M, & de Kloet, E. R. (1990). Mineralocorticoid receptor-mediated changes in membrane properties of rat CA1 pyramidal neurons in vitro. Proceedings of the National Academy of Sciences of the United States of America, 87(12), 4495-4498.

[30] Kable, J. W., & Glimcher, P. W. (2007). The neural correlates of subjective value during intertemporal choice. Nature Neuroscience, 10(12), 1625-1633. doi:10.1038/nn2007

[31] Kahneman, D., & Tversky, A. (1979). Prospect Theory: An Analysis of Decision under Risk. Econometrica, 47(2), 263-291. doi:10.2307/1914185

[32] Karst, H., Berger, S., Turiault, M., Tronche, F., Schutz, G., & Joels, M. (2005). Mineralocorticoid receptors are indispensable for nongenomic modulation of hippocampal glutamate transmission by corticosterone. Proceedings of the National Academy of Sciences of the United States of America, 102, 19204-19207.

[33] Karst, Henk, Berger, S., Erdmann, G., Schütz, G., & Joëls, M. (2010). Metaplasticity of amygdalar responses to the stress hormone corticosterone. Proceedings of the National Academy of Sciences of the United States of America, 107(32), 14449-14454. doi:10.1073/pnas.0914381107

[34] Kőszegi, B., & Rabin, M. (2006). A Model of Reference-Dependent Preferences. The Quarterly Journal of Economics, 121(4), 1133 -1165. doi:10.1093/qje/121.4.1133

[35] Kőszegi, B., & Rabin, M. (2007). Reference-Dependent Risk Attitudes. The American Economic Review, 97(4), 1047-1073. doi:10.1257/000282807783286829

[36] Krawietz, W., Poppert, D., Erdmann, E., Glossmann, H., Struck, C. J., & Konrad, C. (1976). Beta-adrenergic receptors in guinea-pig myocardial tissue. Naunyn-Schmiedeberg's Archives of Pharmacology, 295(3), 215-224.

[37] Laibson, D. (1997). Golden Eggs and Hyperbolic Discounting. The Quarterly Journal of Economics, 112(2), 443 -478. doi:10.1162/003355397555253

[38] Laibson, D., Repetto, A., & Tobacman, J. (1998). Self-Control and Saving for Retirement. Brookings Papers on Economic Activity, 1, 91–196.

[39] Laibson, D., Repetto, A., & Tobacman, J. (2007). Estimating Discount Functions with Consumption Choices over the Lifecycle (Economics Series Working Papers). University of Oxford, Department of Economics. Retrieved from http://ideas.repec.org/p/oxf/wpaper/341.html

[40] Lawrance, E. C. (1991). Poverty and the Rate of Time Preference: Evidence from Panel Data. Journal of Political Economy, 99(1), 54-77.

[41] Leigh, J. P. (1986). Accounting for Tastes: Correlates of Risk and Time Preferences. Journal of Post Keynesian Economics, 9(1), 17-31.

[42] Loewenstein, G. (1996). Out of Control: Visceral Influences on Behavior. Organizational Behavior and Human Decision Processes, 65(3), 272-292.

[43] Loewenstein, G. (2000). Emotions in Economic Theory and Economic Behavior. The American Economic Review, 90(2), 426-432.

[44] Mazur, J. E. (1988). Estimation of indifference points with an adjusting-delay procedure. Journal of the Experimental Analysis of Behavior, 49(1), 37-47. doi:10.1901/jeab.1988.49-37

[45] Murphree, M. (1993). Communal Land Wildlife Resources and Rural District Council Revenues. CASS, University of Zimbabwe, Harare.

[46] Myerson, J., Green, L., Scott Hanson, J., Holt, D. D., & Estle, S. J. (2003). Discounting delayed and probabilistic rewards: Processes and traits. Journal of Economic Psychology, 24(5), 619-635. doi:10.1016/S0167-4870(03)00005-9

[47] Nguyen, Q., & Leung, P. (2010). How Nurture Can Shape Preferences: An Experimental Study on Risk Preferences of Vietnamese Fishers. Environment and Development Economics, 15(05), 609-631. doi:10.1017/S1355770X10000203

[48] Ogaki, M., & Atkeson, A. (1997). Rate Of Time Preference, Intertemporal Elasticity Of Substitution, And Level Of Wealth, 79(4), 564-572.

[49] Pagiola, S. (1996). Price policy and returns to soil conservation in semiarid Kenya, 8(3), 225-271.

[50] Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. Journal of Clinical Psychology, 51(6), 768-774.

[51] Payne, J. W., Laughhunn, D. J., & Crum, R. (1980). Translation of Gambles and Aspiration Level Effects in Risky Choice Behavior. Management Science, 26(10), 1039-1060.

[52] Pender, J. L., & Walker, T. S. (1990). Experimental Measurement of Time Preferences in Rural India (No. 97). ICRISAT, Economics Group Progress Report. Andhra Pradesh: ICRISAT: ICRISAT.

[53] Rachlin, H., Raineri, A., & Cross, D. (1991). Subjective probability and delay. Journal of the Experimental Analysis of Behavior, 55(2), 233-244. doi:10.1901/jeab.1991.55-233

[54] Raeva, D., Mittone, L., & Schwarzbach, J. (2010). Regret now, take it now: On the role of experienced regret on intertemporal choice, 31(4), 634-642.

[55] Richard, T. (1981). Some empirical evidence on dynamic inconsistency. Economics Letters, 8(3), 201-207. doi:10.1016/0165-1765(81)90067-7

[56] Rosenzweig, M. R. (1995). Why Are There Returns to Schooling? The American Economic Review, 85(2), 153-158.

[57] Sarah, L. (1997). The environment and the ethics of discounting: An empirical analysis. Ecological Economics, 20(1), 71-82. doi:10.1016/S0921-8009(96)00073-0

[58] Shamosh, N. A., & Gray, J. R. (2008). Delay discounting and intelligence: A meta-analysis. Intelligence, 36(4), 289-305. doi:10.1016/j.intell.2007.09.004

[59] Shamosh, N. A., Deyoung, C. G., Green, A. E., Reis, D. L., Johnson, M. R., Conway, A. R. A., Engle, R. W., et al. (2008). Individual differences in delay discounting: relation to intelligence, working memory, and anterior prefrontal cortex. Psychological Science, 19(9), 904-911. doi:10.1111/j.1467-9280.2008.02175.x

[60] Shiferaw, B., & Holden, S. T. (2001). Farm-Level Benefits to Investments for Mitigating Land Degradation: Empirical Evidence from Ethiopia. Environment and Development Economics, 6(03), 335-358. doi:10.1017/S1355770X01000195

[61] Slovic, P., Fischhoff, B., & Lichtenstein, S. (1982). Response mode, framing, and information-processing effects in risk assessment. New Directions for Methodology of Social and Behavioral Science: Question Framing and Response Consistency, 21-36.

[62] Stephens, M., & Krupka„ E. L. (2006). Subjective Discount Rates and Household Behavior.

[63] Sullivan, K. A. (2011) The effect of risk, time preference, and poverty on the impacts of forest tenure reform in China. ETD Collection for University of Rhode Island. Paper AAI3464886. http://digitalcommons.uri.edu/dissertations/AAI3464886

[64] Thaler, R. H., & Johnson, E. J. (1990). Gambling with the House Money and Trying to Break Even: The Effects of Prior Outcomes on Risky Choice. Management Science, 36(6), 643-660.

[65] van de Walle, D. (2000). Are returns to investment lower for the poor? Human and physical capital interactions in rural Viet Nam (Policy Research Working Paper Series). The World Bank. Retrieved from http://ideas.repec.org/p/wbk/wbrwps/2425.html

[66] van Stegeren, A., Rohleder, N., Everaerd, W., & Wolf, O. T. (2006). Salivary alpha amylase as marker for adrenergic activity during stress: effect of betablockade. Psychoneuroendocrinology, 31(1), 137-141. doi:10.1016/j.psyneuen.2005.05.012

[67] Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. Journal of Personality and Social Psychology, 54(6), 1063-1070.

Chapter 7

Bi-directional Effect of Stress on Present Bias in Intertemporal Choice

7.1 Summary

Intertemporal choices, involving decisions which trade off instant and delayed outcomes, are often made under stress. It remains unknown, however, how stress affects intertemporal choice. Here we use theory and evidence from behavioral economics and cellular neuroscience to demonstrate a bi-directional effect of stress on intertemporal choice. First, we distinguish between two distinct motives within intertemporal choice, i.e. present bias and impatience, and show that stress affects the former, but not the latter. Second, consistent with evidence from cellular neurobiology, we show that present bias is increased immediately after stress, but decreased when subjects are tested 20 minutes later. Furthermore, the degree to which individual participants show these opposite effects of stress on present bias correlates strongly with individual cortisol changes in response to stress, suggesting that cortisol may drive these behavioral effects. Thus, deferring an economic decision after stress can have a significant impact on the outcome.

7.2 Introduction

Many everyday decisions entail trading off immediate and delayed outcomes. In such intertemporal choices, both humans and animals tend to attach special significance to short-term rewards, a phenomenon known as "present

bias" (1-4). As a consequence of present bias, people frequently find it difficult to act in accordance with their own long-term interest (4, 5).

Intertemporal choices in private or professional contexts are often made under stress; managers, politicians, investment bankers, medical doctors and other professionals make vital decisions under a considerable amount of pressure. This applies, for instance, to a corporate executive who needs to trade-off the usefulness of long-term business strategies with succumbing to the pressure of reporting short-term profits, or a medical doctor who needs to decide on the spot between quick fixes relieving the symptoms of his patients, or slower therapies. How stress affects intertemporal choice, though, is unknown. Here we combine theory and evidence from behavioral economics and cellular neuroscience to answer this question.

In behavioral economics, both models and evidence on intertemporal choice now distinguish between present-bias on the one hand, and impatience on the other (4). While impatience is simply the subject's preference about consumption at different times (i.e. as soon as possible in case of high impatience), present bias is normatively irrational because it leads subjects to fail executing the future plans that they make today. Here we econometrically and experimentally distinguish between these two motives, and can thus ask whether stress differentially affects present bias or impatience.

In cellular and behavioral neuroscience, it has become evident in recent years that stress affects neurobiological processes and cognitive function in two distinct temporal domains (6-8). Broadly, the picture that emerges from this research is that immediately after stress, the stress-induced changes in hormone and neurotransmitter levels facilitate short-term solutions to the stressful situation; in contrast, beginning approximately 1 h after stress onset, slower physiological changes promote restoration and long-term protection after stress. More specifically, shortly after stress, corticosteroid hormones and noradrenaline synergistically promote rapid increases in neuronal activity, such as activity caused by the neurotransmitter glutamate (9, 10), the main excitatory neurotransmitter in the nervous system. Rapid effects of corticosteroids have been described for several emotion- and arousal-related brain regions such as the hippocampus, amygdala, and medial prefrontal cortex (11). These early physiological responses to stress likely facilitate the rapid focused attention, hypervigilance and choice of strategy required to implement the organism's fight-or-flight response (6); in particular, they promote habitual, reflex-like behavior at the expense of goal-directed behavior (12).

In contrast, the slow actions of cortisol focus on long-term restoration and protection after stress. Through changes in gene transcription that require 55-65 minutes to develop and last for several hours (13), stress-induced corticosteroid actions shut down the effects of noradrenaline (14-16) and change

neuronal activity in frontal brain regions such that the stress-induced release of hormones from the pituitary is terminated (17, 18). Behaviorally, these delayed effects of stress promote consolidation of stress-related memories for future use (6, 19) and stimulate restoration of cognitive self-control (7, 20, 21). Although these slow corticosteroid actions develop within an hour, their implications stretch well beyond this time-domain (22, 23).

We hypothesized that this bi-directional pattern is also apparent in more complex behavioral responses carried out after stress, such as intertemporal choice. Because the early physiological responses facilitate an increased focus on the here and now, we expect to see stronger present bias immediately after stress. By contrast, because the restorative functions of the slower actions of stress hormones that prepare the organism for the future engage dorsolateral prefrontal networks, which are also recruited during self-controlled, far-sighted decision making (24-26), we expected to find less present-bias (cf. longer time-horizons) in the recovery phase some time after stress.

7.3 Results

To test this hypothesis, 81 healthy male participants were randomly assigned to one of four conditions. The experimental groups did neither differ in age, BMI nor on the Barratt Impulsivity Scale (P's > 0.1). Half of the participants were subjected to a group-wise Trier Social Stress Test (TSST-G; 27, 28), in which a maximum of 4 individuals sequentially had to deliver a public speech and perform mental arithmetic in front of a non-responsive panel and a video camera (Materials and Methods). The subjects in the control groups underwent the exact same conditions, except that they all delivered their speech and performed arithmetic simultaneously with one another and without active observers; this removed the stressful component of social control, while holding cognitive load constant as much as possible. Salivary cortisol and alpha-amylase (sAA; an index of adrenergic activity; 29) levels were measured at several time points during the experiment as an index of the individual's stress responsivity. Subsequently, individuals of both the control and stress groups performed an intertemporal choice task, in which they made a series of choices between sooner-smaller and larger-later rewards with varying delays (Materials and Methods). Critically, half of the subjects in the stress and control groups performed this task directly after stress exposure to target rapid nongenomic actions of stress hormones (early groups), while the other half performed the task 20 minutes later (late groups), i.e. between 55 and 65 min after onset of the stressor. The latter time-point was selected such that it would just allow the development of

genomic actions in the PFC (approximately one hour after onset of the TSST-G; 16, 17) while noradrenergic activity is restored, and yet be as close as possible to the earlier time-point (a 20 minutes difference), to avoid unwanted influences that could not be controlled for such as circadian variations in hormone release.

7.3.1 Stress Induction

The experimental groups did not differ on any of the measured baseline variables (cortisol, PANAS and VAS; all P's > 0.40). As expected, the ANOVA for cortisol showed a significant Sample Period x Stress interaction (Figure 7.3.1a, $F_{1.6,124.4} = 34.70$, $P < 0.001$). Furthermore, a main effect of Sample Period ($F_{1.6,124.4} = 34.02$, $P < 0.001$) and a main effect of Stress ($F_{1,77} = 38.53$, $P < 0.001$) were found. Planned simple contrasts related to baseline showed that subjects in the stress condition had increased cortisol levels from 30 minutes after TSST-G onset (t30) until t90, i.e. at the end of the session (all P's < 0.001).

For sAA, a significant Sample Period x Stress interaction ($F_{5.1,395}$=8.89, P<0.001, Fig. 1b) and a significant main effect of Sample Period ($F_{5.1,395}$=23.12, P<0.001) were found. Planned simple contrasts compared to baseline showed that sAA levels were increased in the stress condition from t30, i.e. during the TSST-G, until after the early test condition at t50 (P's<0.05). Negative affect (PANAS; $F_{1,77} = 15.60$, $P < 0.001$) as well as subjective stress ratings (VAS; $F_{1,77} = 15.82$, $P < 0.001$) both increased in the stress group compared to the control group immediately after the stress task compared to baseline. As expected, no effects of Timing (Early vs. Late groups) were found on the stress induction measurements.

7.3.2 Intertemporal Choice Performance

In intertemporal choice, present bias can be discerned from impatience: whereas the latter refers to the degree of discounting of future outcomes as a function of time, the former refers to extra value placed on short-term outcomes (4). We dissociated the effects of stress on present bias and impatience by characterizing our participants' choices over time with Laibson's (4) quasi-hyperbolic discounting model, in which present bias is characterized by the beta parameter, and impatience by the delta parameter. Strong present bias and strong impatience are associated with low betas and high deltas, respectively (Materials and Methods).

We found that stress differentially affects present bias, but not impatience, depending on the time-of-testing after stress. Specifically, we ob-

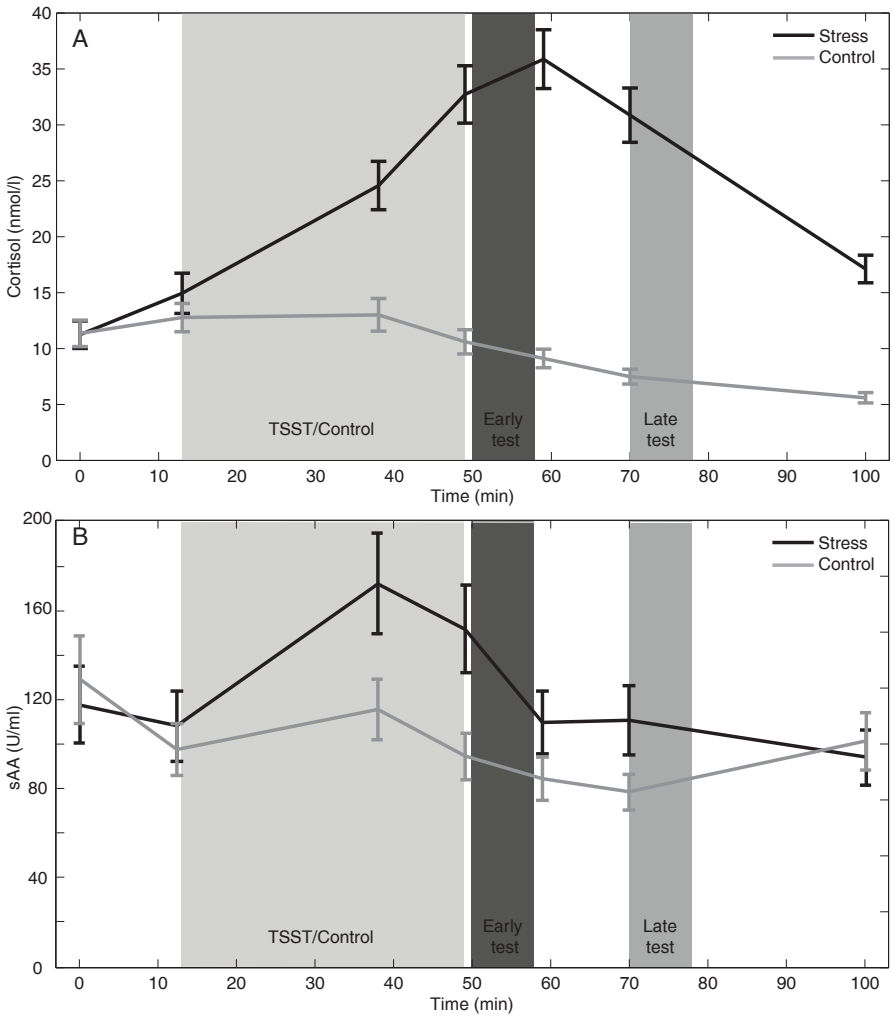

Figure 7.3.1: Experiment timeline and evolution of cortisol (a) and alpha-amylase (b) levels. Subjects were subjected to a group-wise Trier Social Stress Test (TSST-G) or a control task, which lasted 20 minutes (light gray region, "TSST/Control"). Subsequently, they performed an intertemporal choice task lasting approximately 8 min, either immediately following the TSST-G/Control task (dark gray region, "Early test"), or 20 min later i.e. 55 min after onset of the stressful situation (medium gray region, "Late test"). Salivary cortisol (a) and alpha-amylase (sAA) (b) levels were measured throughout the experimental session and are shown separately for the stress groups (black line) and control groups (gray line). Error bars indicate one SEM.

served a two-way stress x time interaction on the beta parameter with age and BMI included as covariates ($F_{2,67} = 4.12$, $P = 0.02$ (two-tailed)). Planned comparisons revealed that when tested immediately after stress, cortisol responders to stress (see SI Materials and Methods for definition) exhibited stronger present bias than controls ($F_{1,32} = 4.46$, $P = 0.02$ (one-tailed) / $P = 0.04$ (two-tailed); Figure 7.3.2a). By contrast, individuals tested at the later time-point were less present-biased than controls ($F_{1,34} = 5.63$, $P = 0.01$ (one-tailed) / $P = 0.02$ (two-tailed)). Similar results were obtained when age and BMI were not included as covariates (stress x delay interaction: $F_{1,70} = 7.45$, $P = 0.008$ (two-tailed); early stress vs. control: $F_{1,36} = 5.10$, $P = 0.003$ (two-tailed)/ $P = 0.002$ (one-tailed); late stress vs. control: $F_{1,34} = 2.75$, $P = 0.107$ (two-tailed) / $P = 0.053$ (one-tailed)), or when non-responders were included in the data pool (see SI Results). To further corroborate this finding, we regressed the probability of being a cortisol non-responder to stress on a number of observable variables and found that non-responders are similar to responders on important variables, and show that their exclusion is unlikely to have introduced significant selection bias (see SI Results).

No significant effects of stress and timing were found on impatience (delta) (interaction: $F_{1,68} = -0.09$, $P = 0.77$; early stress vs. control: $F_{1,32} = 0.02$, $P=0.88$; late stress vs. control: $F_{1,34} = 1.39$, $P = 0.25$; Figure 7.3.2b).

How large are these effects of stress on present-bias in real-life terms? The beta-parameter cannot be translated into an implied interest rate, since it represents the same decrease of subjective value for all delayed outcomes, while interest rates accrue exponentially. However, we can ask how particular choices a subject might be faced with would be affected by stress. Consider a decision between receiving CHF 40 in 3 months and 1 day vs. receiving a smaller amount tomorrow. In the control condition, beta = 0.82; with this degree of present bias, subjects will just accept CHF 32.80 tomorrow instead of CHF 40 in 3 months and 1 day. In the early condition, beta changes to 0.75; this implies that the same subject would now accept CHF 30 tomorrow instead of CHF 40 in 3 months and 1 day. Conversely, in the late condition, beta changes to 0.92; this implies that the same subject would require CHF 36.80 tomorrow to forgo CHF 40 in 3 months and 1 day Together, the effects of stress on present bias span 17 percentage points of the amount in question, which is a large effect.

We opted for Laibson's quasi-hyperbolic model to characterize our subjects' choices because it allows to dissociate present-bias from impatience, but also because our data were better described by the quasi-hyperbolic model than the standard hyperbolic discounting model or the standard exponential model (Akaike Information Criteria: Laibson model, -11.80; hyperbolic model, -9.22; exponential model, -8.46). Nevertheless, to support our find-

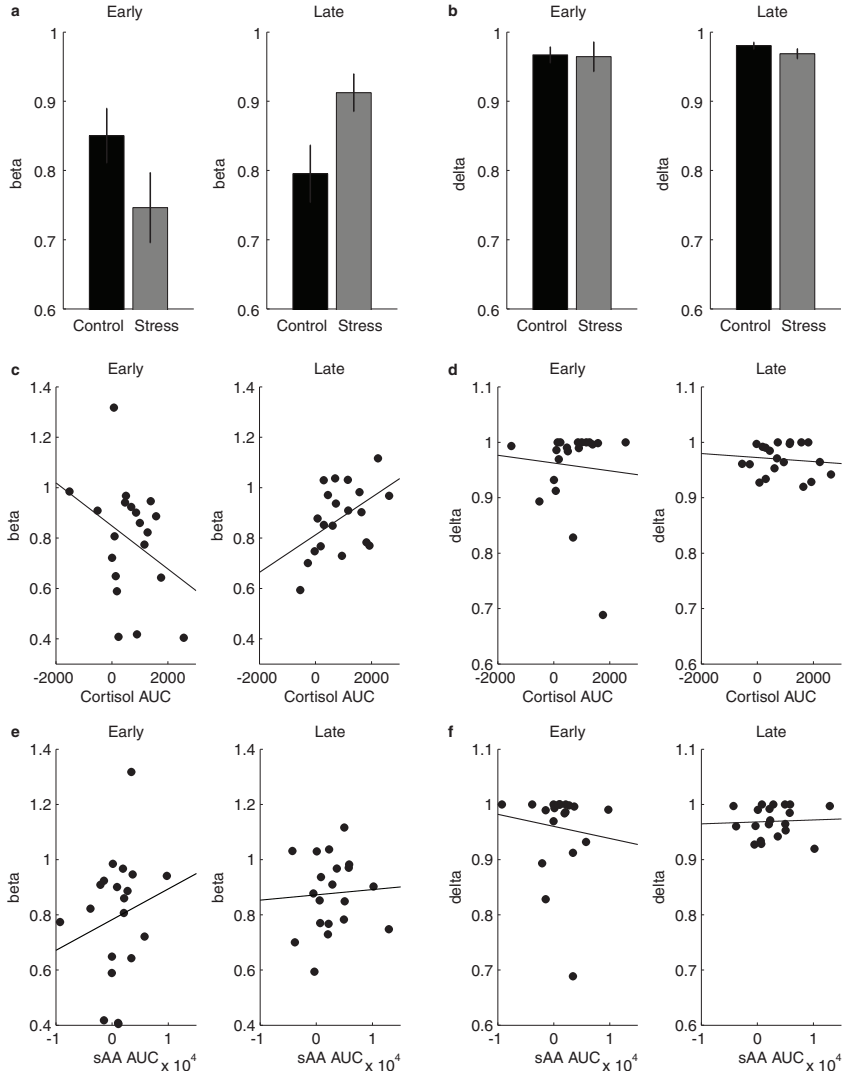

Figure 7.3.2: Effect of stress on time preference. a) The present bias parameter beta was lower in the stress than the control group immediately after stress ("Early"). Since lower betas reflect increased present bias, this finding suggests that the immediate effect of stress was to increase present bias in intertemporal choice. In contrast, beta was higher in the stress than in the control group when tested 20 min later ("Late"), suggesting reduced present bias. b) The impatience parameter delta was not affected by stress either during early or late testing. c) Scatter plot representing individual stress responsivity to the TSST-G in the stress groups, measured by area-under-the-curve (AUC) for salivary cortisol. Individuals in the stress groups with higher stress responsivity showed a larger stress effect on present bias. Crucially, this effect went in opposite directions in the early and late groups: high stress responsivity predicted lower beta (higher present bias) in the early stress group (slope coefficient: t_{19} = -1.95, P = 0.03), while it predicted higher beta (reduced present bias) in the late stress group (t_{19} = 2.24, P = 0.02). d) Individual cortisol responsivity to stress did not predict changes in the delta (impatience) parameter (early stress group: t_{19} = -0.30, P = 0.77; late: t_{19} = -0.52, P = 0.61).

ings, we also fit the standard hyperbolic model (30) and the standard exponential model. The results were comparable to those obtained for the beta parameter in Laibson's quasi-hyperbolic model (see SI Results).

We next tested whether this bi-directional effect of stress on present bias might be associated with stress responsivity, here probed by the stress-induced increases in salivary cortisol and sAA levels (area-under-the-curve, AUC; SI Materials and Methods). To this end, we used regression to predict the beta parameters from the stress-induced cortisol and sAA responses of individuals in the early or late groups. Indeed, the individual betas were predicted by cortisol AUC, and crucially, the sign of this effect differed as a function of time-of-testing after stress: immediately after stress, high cortisol responses predicted greater present bias (lower betas; significance of coefficient on cortisol AUC: t_{19} = -1.95, P = 0.03; Figure 7.3.2c), whereas later on, high cortisol responses predicted decreased present bias (higher betas; t_{19} = 2.24, P = 0.02). Correlations in the same directions were also observed when considering only cortisol measurements until the time of the intertemporal choice task for calculating area under the curve (see SI Results). However, no association was found between changes in present bias and stress-induced sAA increases (significance of coefficient on sAA area-under-the-curve, early: t=1.136, P=0.189; late: t=0.23, P=0.817). Thus, the bi-directional effect of stress on intertemporal choice might be driven specifically by differential time-dependent effects of cortisol. Interestingly, present-bias was nearly indistinguishable for the early and late groups at the intersect of the beta-trendline and cortisol AUC = 0 (i.e. no rise in salivary cortisol level in response to the Trier Social Stress Test), further strengthening a causative role of corticosteroid hormones in the bi-directional effect. The impatience parameter delta was not predicted by cortisol increases (early stress group: t_{19} = -0.30, P = 0.77; late: t_{19} = -0.52, P = 0.61; Figure 7.3.2d).

A possible confound to our results is that subjects' responses to the time preference questions may be inconsistent, and that this inconsistency may be modulated by stress. However, additional analyses showed that this appeared not to be the case (see SI Results).

A further potential concern is that the responses in the intertemporal choice task themselves may have influenced cortisol levels; in particular, in the early group, subjects may have made particular choices to regulate their own mood after stress induction. However, the mood changes induced by the stress task, if anything, counterveiled the relationship between cortisol and present bias in the stress group (for details and additional analyses see SI Results). Mood regulation is therefore unlikely to be the underlying driver of our results. We also did not find any evidence that cortisol levels differ between the early and late groups (see SI Results), rendering it unlikely that

choices in the time preference task affected cortisol levels.

7.4 Discussion

We here demonstrate two dissociations in the effect of stress on intertemporal choice: stress affects present bias, but not impatience; second, stress increases present bias immediately after stress, but decreases it when subjects are tested 20 minutes later, consistent with evidence from cellular neurobiology. In addition, we supply evidence for the biological mechanism underlying our effects: the degree to which individual participants show these opposite effects of stress on decision-making correlates strongly with individual cortisol responsivity to stress.

This bi-directional effect of stress on present bias is in line with current views that shortly after stress individuals turn to simple behavioral strategies. For instance, humans exposed to a psychosocial stressor use a simpler (striatal) stimulus-response rather than a more complex spatial learning strategy (31). Individuals also shift from goal-directed to habitual control in instrumental behavior shortly after stress (12). Underlying biological mechanisms of these rapid stress effects are thought to involve both catecholamines and corticosteroid hormones (7, 8, 12, 31), the latter probably accomplishing non-genomic actions (9). Our results show that higher individual cortisol responses predicted greater present bias, while no associations were found with the noradrenergic marker alpha-amylase (sAA). Whether the rapid behavioral effects involving the prefrontal-striatal circuitry are due to non-genomic corticosteroid cellular actions –similar to those described e.g. for hippocampal neurons (9)- is presently unknown. Furthermore, sAA levels correlate with peripheral adrenaline levels, which may not entirely reflect (nor)adrenaline levels in the brain, despite the fact that adrenaline can indirectly cause release of noradrenaline in the brain (32). Moreover, we cannot exclude that other stress-induced factors, such as the peptide corticotrophin releasing hormone, may play a role in intertemporal choices. The later effects of stress may serve the function of normalizing the stress response and preparing the organism for the future (7, 20, 21); consistent with this view is the finding that slow genomic corticosteroid effects induced by stress improve spatial memory formation in mice (19). At this stage we can only speculate about a role of glucocorticoid receptors in this phase; these receptors play a prominent role in the delayed restorative capacity after stress (6, 19) including in the prefrontal cortex (17, 18, 20, 21) but the 55 minute delay between onset of the stressor and behavioral measurements is relatively short (though not unprecedented; 33) for genomic actions to take place. Clearly,

future studies will need to vary the delay between stress onset and task performance in order to support a role of long-term, gene-mediated glucocorticoid actions.

An intriguing facet of our findings is that we found a dissociation between stress effects on present bias and impatience. What might be the reason for this finding? One possibility is that impatience is a stable trait, while present bias, although correlated within subjects over time, is more susceptible to situational influences; in particular, it is conceivable that present bias reflects the need of escaping a potentially threatening situation, and that therefore it is modulated by short-term fluctuations in stress levels.

Our results complement and extend several previous studies of the effect of stress on decision-making. Keinan (34) found that subjects were impaired in a verbal analogy task when they were threatened with uncontrollable compared to controllable electric shocks. Gray (35) found that subjects made suboptimal decisions in a temporally extended choice task when the task was presented in a negative emotional compared to a neutral context. Van den Bos et al. (36) and Preston et al. (37) found that performance on the Iowa Gambling Task was impaired under stress, particularly in men. Finally, Porcelli & Delgado (38) induced stress using the cold-pressor task, in which subjects immerse their hand in ice-cold water, and found that this stress induction increased the reflection effect in risky decision-making: stressed subjects showed stronger risk aversion in the gains domain, and stronger risk seeking in the loss domain. Our study extends these previous findings on risky choice into the intertemporal domain; to our knowledge, our experiment is the first to test the effect of stress relative to intertemporal choice, and its dependency on the timing of choice relative to stress onset. Notably, we report here on differences in present bias for future rewards after two delays: directly after a brief period of stress, when both corticosteroids and noradrenergic mechanisms play a role, and 20 minutes later, i.e. 55 minutes after individuals start to be exposed to stress, allowing genomic actions of corticosteroid hormones, while keeping other factors as constant as possible. Furthermore, in the current study the different delays of reward choices were limited, with the soonest reward delay being tomorrow (and, for practical reasons, not a more present choice like today). Future studies will need to explore different time scales, varying the delay between stress onset and the task as well as reward delays within the intertemporal choice task, to fully understand the complexity of the effects of stress and stress hormones on intertemporal choice. In general, our findings suggest that time preference is not a stable trait, as traditionally assumed in economic theory (39, 40) but is strongly susceptible to environmental and somatic factors, such as individuals' responses to stress and variations in hormonal balance. Regardless

of the internal mechanisms, the fragility of time preference and its complex dependence on stress need to be considered in the design of optimal policies aiming at decisions that are consistent with an individual's long-term economic interests.

7.5 Materials and Methods

7.5.1 Stress manipulation

81 male undergraduate students from the University of Zurich ranging in age from 19 to 30 (M = 21.31 ± 1.85) participated in the study (for details of the criteria see SI Material & Methods).

Psychosocial stress was induced with a grouped version of the Trier Social Stress Test (TSST-G; 27, 28). The procedure followed closely that described by Dawans et al. (28) and involved a preparation period of 5 min, followed by a video- and audio- taped public speaking task of 12 min (a fictional job interview, see below), and a mental arithmetic task of 8 min, both in front of an evaluation committee (one man and one woman wearing white laboratory coats). A maximum of 4 and a minimum of 2 subjects were tested at the same time. The subjects in the control groups underwent the exact same conditions, except that they all delivered their speech and performed arithmetic simultaneously with one another and without active observers; this removed the stressful component of social control, while holding cognitive load constant. A more detailed description of the TSST-G and the control task can be found in the SI Materials and Methods.

7.5.2 Salivary Sampling and Biochemical Analysis

Salivary samples were obtained using Salivette sampling devices (Sarstedt, Nümbrecht, Germany) at 7 time points during the experiments (Figure 7.3.1). Salivary samples were stored at -20°C until further analysis. Free cortisol levels were measured using a commercially available immunoassay (IBL, Hamburg, Germany). Salivary alpha-amylase (sAA) levels were measured by a quantitative enzyme kinetic essay as described elsewhere (29).

7.5.3 Questionnaires

Mood measurements and stress ratings were assessed shortly before and directly after the TSST-G or control task (at t=15 and t=50 min, see Figure 7.3.1). Subjects filled out the 10 negative affect items (rated on a 5-point

scale) of the Positive and Negative Affect Scale (PANAS; 41), resulting in a score of negative affect before and after the TSST-G or control task. At the same time points they rated how stressed they felt at that moment on a Visual Analogue Scale (later coded as ranging from 1 to 100). To assess impulsivity as a personality trait (42), subjects filled out the 30-item Barratt Impulsivity Scale (BIS) after the experimental tasks.

7.5.4 Intertemporal Choice Task

Participants performed 6 blocks of an intertemporal choice task with varying delays, where decisions between a sooner smaller reward and a later larger reward were offered. In the first four blocks subjects had the choice between a smaller reward tomorrow, and a larger reward in a) 3 months and 1 day, b) 6 months and 1 day, c) 9 months and 1 day, and d) 12 months and 1 day. The short delay was set to "tomorrow" rather than "today" to keep transaction costs the same for sooner and later payments. In the last two blocks, subjects chose between a smaller reward in 6 months and 1 day, and a larger reward in e) 9 months and 1 day, and f) 12 months and 1 day. Each block consisted of 7 binary choice trials, resulting in a total of 42 trials. The larger reward was kept constant at an amount of 40 Swiss Francs (CHF), while the sooner smaller reward started at CHF 20 and was then adjusted with a titration method according to the choices the subject made (for details on the titration method and calculation of the indifference points see SI Materials and Methods).

Reimbursement consisted of a flat rate of CHF 10 and a variable payment depending on participants' choices. In particular, as was explained to the participants at the beginning of the study, one of all the choices made was randomly selected at the end of the study, and the chosen option on this trial was paid out for real, i.e., participants could pick up the chosen amount on the chosen day of delivery, using a voucher valid at the University cashier's office. As mentioned, transaction costs were kept constant by setting the soonest outcome to "tomorrow".

7.5.5 Procedure

Subjects were randomly assigned to one of four conditions: control-early, control-late, stress-early or stress-late. Three groups contained 20 participants and one group (control-late) 21 participants. The study was conducted between 14:00 and 20:00 in the (late) afternoon, when plasma cortisol levels are close to the circadian trough. Participants were instructed to refrain from smoking, eating, or drinking caffeine containing beverages at least 2h before

the study, and were asked not to consume alcohol 24h before participating. An overview of the study timeline is displayed in Figure 7.3.1. Subjects were instructed not to talk to each other during the whole experiment.

Twenty minutes after subjects arrived in the laboratory, a first saliva sample was taken. Subjects were guided to a room where they received instructions and practice questions for the intertemporal choice task, to be able to administer the task directly after the stress situation without delay. Notably, subjects only received general instructions, but were not provided with any information about the actual rewards and delays during the intertemporal choice task. It is therefore unlikely that they would have decided on their choices already at this timepoint. When all subjects had understood the task and answered the practice questions correctly, a second saliva sample was taken and a PANAS/VAS questionnaire was filled out. Next, subjects received instructions for the TSST-G or the control task, and after the 5 min preparation period participants were guided to another room, where they gave their speech. Before subjects were instructed to perform the arithmetic task, a third saliva sample was taken. Directly after the whole TSST-G or control procedure, a fourth saliva sample was taken and a further PANAS/-VAS questionnaire was filled out. Next, participants were asked to sit at the chair placed behind them and, depending on the experimental condition, directly perform the intertemporal choice task on a laptop placed before them on a table (the early groups), or fill out the Barratt and a socioeconomic questionnaire and read neutral magazines (the late groups). The total delay between the start of the TSST-G and testing of intertemporal choice in the early groups including transportation time from one room to another, biological measurements and filling out forms was 35 minutes. 10 min and 20 min after the end of the TSST-G or control task, the fifth and sixth saliva samples were taken, after which subjects in the late groups performed the intertemporal choice task (20 minutes after the early groups), and subjects in the early groups filled out the questionnaires and read magazines. After they finished, participants waited until the last saliva sample was taken 50 min after the end of the TSST-G or control task, after which they were debriefed and got their payment results (depending on the choices they made during the intertemporal decision making task), and instructions for picking up their payment.

The choice of timing of the behavioral tasks after the stress task was based on the following reasoning. The first time-point was selected to target nongenomic actions of corticosteroid hormones and other rapidly acting stress hormones like noradrenaline, i.e. immediately after the TSST. At this point in time, levels of the stress hormones (including of (nor)adrenaline) are still high, so that they can evoke non-genomic actions (9, 10); however, the

time-frame is too short to allow the development of gene-mediated events. The second time-point was selected such that it would just allow the development of genomic actions. Specifically, earlier findings in neurobiology show that genomic corticosteroid actions are apparent after one hour, both in the hippocampus (see e.g. 43) and the prefrontal cortex (17). However, we wished to not test later than approximately one hour after stress onset in order to be as close as possible to the earlier time-point, to avoid unwanted influences that cannot be controlled for, such as circadian variations in hormone level. For this reason we tested individuals between 55 and 65 minutes after onset of the TSST.

7.5.6 Model Fits

For every subject and every delay level, we determined the amount at which a subject was indifferent between the immediate and delayed option (in items with an immediate option), or the delayed and the very delayed option (in items with an added front-end delay) based on the individual switching points (see above). This allowed us to express the subjective value of the delayed reward as a fraction of the subjective value of the immediate reward. We then plotted the relative values of the delayed rewards as a function of time. Next, for every subject, we fitted three different models to the obtained indifference points. The hyperbolic discounting model and the exponential discounting model are described in the SI Materials and Methods.

Beta-delta quasi-hyperbolic discount model. Laibson's beta-delta model (4) was fitted to the indifference points to obtain an estimate of the degree of impatience and present bias:

$$V_t = v\left(r_t\right) + \sum_{\tau=1}^{T-t} \delta_t v\left(r_{t+\tau}\right),$$

where V_t indicates the discounted value at time t of a stream of rewards r with subjective values v as a function of time τ. This equation contains a constant, exponential discount function whose discount rate is $\ln \frac{1}{\delta}$, thus whose steepness can be characterized by δ. The β parameter deflects the exponential discount curve and its inverse can be interpreted as the extra weight added to immediate rewards. Hence, δ can be interpreted as measuring impatience and β as measuring present bias.

7.5.7 Statistical Analysis

All measures that showed a skewed distribution with the Shapiro-Wilk test of normality (cortisol, sAA, PANAS and VAS measurements) were log-transformed and further Analysis of Variance (ANOVAs) were performed on the transformed data. Hormone measurements were analyzed using a 7 (Sample Period: t0 vs. t15 vs. t40 vs. t50 vs. t60 vs. t70 vs. t100) x2 (Stress: TSST-G vs. Control) x2 (Timing: Early vs. Late) General Linear Model (GLM) repeated measures ANOVA with Sample Period as a repeated measure. To assess if choices in the time preference task might have affected cortisol levels, the above GLM repeated measures ANOVA was performed separately for cortisol measurements in the stress and control groups, with Timing (Early vs. Late) as a between-subjects factor. Subjective affect and stress measures were analyzed using a 2 (Sample Period: pre vs. post) x2 (Stress: TSST-G vs. Control) x2 (Timing: Early vs. Late) General Linear Model (GLM) repeated measures ANOVA with Sample Period as a repeated measure.

For our main analysis, performance on the intertemporal choice task was analyzed by testing the model parameters with a 2 (Stress: TSST-G vs. Control) x 2 (Timing: Early vs. Late) MANOVA with age and body mass index (BMI) as covariates (for details see SI Methods). Results without covariates are reported in the Results section.

In case of significant main effects (to assess the stress manipulation) and interaction effects (to assess the effects of stress and timing on delay discounting) planned comparisons were performed. Since we had specific hypotheses about the direction of the behavioral effects at the two different delays, one-tailed tests are justified here; in particular, previous literature predicted that immediately after stress, present bias would be increased, while later it would be decreased. Nevertheless, our main results remain significant when performing two-tailed hypothesis tests, as reported in the Results section.

The relationship between hormone increases and stress-induced changes in discount parameters was assessed by OLS regression of model parameters on area-under-the-curve for cortisol and sAA using heteroskedasticity-robust standard errors (for details see SI Methods).

Bibliography

[1] Benzion U, Rapoport A, & Yagil J (1989) Management Science 35, 270-284.

[2] Frederick S, Loewenstein G, & O'Donoghue T (2002) Journal of Economic Literature 40, 351-401.

[3] Kalenscher T & Pennartz CM (2008) Prog Neurobiol 84, 284-315.

[4] Laibson D (1997) Quarterly Journal of Economics 112, 443-477.

[5] Haynes L (2009) Postnote 328.

[6] de Kloet ER, Joels M, & Holsboer F (2005) Nat Rev Neurosci 6, 463-475.

[7] Joels M, Fernandez G, & Roozendaal B (2011) Trends Cogn Sci 15, 280-288.

[8] Schwabe L, Wolf OT, & Oitzl MS (2010) Neurosci Biobehav Rev 34, 584-591.

[9] Karst H, Berger S, Turiault M, Tronche F, Schutz G, & Joels M (2005) Proc Natl Acad Sci U S A 102, 19204-19207.

[10] Karst H, Berger S, Erdmann G, Schutz G, & Joels M (2010) Proc Natl Acad Sci U S A 107, 14449-14454.

[11] van Marle HJ, Hermans EJ, Qin S, & Fernandez G (2010) Neuroimage 53, 348-354.

[12] Schwabe L, Tegenthoff M, Hoffken O, & Wolf OT (2010) J Neurosci 30, 8190-8196.

[13] Datson NA, Morsink MC, Meijer OC, & de Kloet ER (2008) Eur J Pharmacol 583, 272-289.

[14] Pu Z, Krugers HJ, & Joels M (2007) Learn Mem 14, 359-367.

[15] Pu Z, Krugers HJ, & Joels M (2009) Learn Mem 16, 155-160.

[16] Joels M & de Kloet ER (1989) Science 245, 1502-1505.

[17] Hill MN, McLaughlin RJ, Pan B, Fitzgerald ML, Roberts CJ, Lee TT, Karatsoreos IN, Mackie K, Viau V, Pickel VM, et al. (2011) J Neurosci 31, 10506-10515.

[18] Yuen EY, Liu W, Karatsoreos IN, Feng J, McEwen BS, & Yan Z (2009) Proc Natl Acad Sci U S A 106, 14075-14079.

[19] Oitzl MS, Reichardt HM, Joels M, & de Kloet ER (2001) Proc Natl Acad Sci U S A 98, 12790-12795.

[20] Henckens MJ, van Wingen GA, Joels M, & Fernandez G (2010) J Neurosci 30, 12725-12732.

[21] Henckens MJ, van Wingen GA, Joels M, & Fernandez G (2011) Proc Natl Acad Sci U S A 108, 5801-5806.

[22] van Stegeren AH, Roozendaal B, Kindt M, Wolf OT, & Joels M (2010) Neurobiol Learn Mem 93, 56-65.

[23] Cornelisse S, van Stegeren AH, & Joels M (2011) Psychoneuroendocrinology 36, 569-578.

[24] Figner B, Knoch D, Johnson EJ, Krosch AR, Lisanby SH, Fehr E, & Weber EU (2010) Nat Neurosci 13, 538-539.

[25] Hare TA, Camerer CF, & Rangel A (2009) Science 324, 646-648.

[26] McClure SM, Laibson DI, Loewenstein G, & Cohen JD (2004) Science 306, 503 - 507.

[27] Kirschbaum C, Pirke KM, & Hellhammer DH (1993) Neuropsychobiology 28, 76-81.

[28] von Dawans B, Kirschbaum C, & Heinrichs M (2011) Psychoneuroendocrinology 36, 514-522.

[29] van Stegeren A, Rohleder N, Everaerd W, & Wolf OT (2006) Psychoneuroendocrinology 31, 137-141.

[30] Mazur JE (1984) Journal of Experimental Psychology: Animal Behavior Processes 10, 426-436.

[31] Schwabe L, Oitzl MS, Philippsen C, Richter S, Bohringer A, Wippich W, & Schachinger H (2007) Learn Mem 14, 109-116.

[32] Williams CL & McGaugh JL (1993) Behav Neurosci 107, 955-962.

[33] Sapolsky RM, Romero LM, & Munck AU (2000) Endocr Rev 21, 55-89.

[34] Keinan G (1987) J Pers Soc Psychol 52, 639-644.

[35] Gray JR (1999) Pers Soc Psychol Bull 25, 65-75.

[36] van den Bos R, Harteveld M, & Stoop H (2009) Psychoneuroendocrinology.

[37] Preston SD, Buchanan TW, Stansfield RB, & Bechara A (2007) Behav Neurosci 121, 257-263.

[38] Porcelli AJ & Delgado MR (2009) Psychol Sci 20, 278-283.

[39] Samuelson P (1937) Rev. Econ. Stud. 4, 155–161

[40] Lancaster K (1963) International Economic Review 4, 221-231.

[41] Watson D, Clark LA, & Tellegen A (1988) J Pers Soc Psychol 54, 1063-1070.

[42] Patton JH, Stanford MS, & Barratt ES (1995) J Clin Psychol 51, 768-774.

[43] Joels M & de Kloet ER (1990) Proc Natl Acad Sci U S A 87, 4495-4498.

7.6 Supporting Materials and Methods

7.6.1 Selection of subjects

We restricted our experiment to men since controlling for ovarian cycle in women is logistically difficult. Before admission to the study, all subjects were screened in a telephone interview to exclude medication intake, somatic diseases, or any neurological or psychiatric disorders. Furthermore, psychology and economics students, self-reported heavy smokers (consumption of $>$ 5 cigarettes per day), heavy alcohol consumers (consumption of $>$ 60 g alcohol per day) and drug users were excluded. Participants were German native speakers, had not participated in a Trier Social Stress Test (TSST) before and would stay in Zurich at least for the next 12 months (for payment of their reward). The study was approved by the ethical committee of the University of Zurich and all participants provided written informed consent. Participants received a variable reimbursement for their participation, depending on the choices they made during the experiment.

7.6.2 Stress Manipulation

Psychosocial stress was induced with a grouped version of the Trier Social Stress Test (TSST-G; (1, 2). The procedure followed closely that described by Dawans et al.(2) and involved a preparation period of 5 min, followed by a video- and audio- taped public speaking task of 12 min (a fictional job interview, see below), and a mental arithmetic task of 8 min, both in front of an evaluation committee (one man and one woman wearing white laboratory coats). A maximum of 4 and a minimum of 2 subjects were tested at the same time. In the job interview component of the task, participants had 3 minutes to describe why their personal qualities qualified them for a job. The committee repeatedly interrupted the presentation with questions, following a pre-prepared script. In the arithmetic task, participants were asked to count backwards in steps of 16, starting at a random 4-digit number. When a mistake was made the panel told the participant to start over. Subjects all delivered their speech and after that performed the arithmetic task. Each subject was called at least twice and in random order for every task, to induce a feeling of unpredictability. Speaking time for every participant was kept constant.

To keep the cognitive load and circumstances of the control condition as comparable as possible, only lacking the component of social control, subjects in the control condition underwent the same conditions, with three important differences. First, subjects were not video- or audio- taped and there was no

panel in laboratory coats, just a passive observer in a corner of the room. Second, the public speech was replaced by an account of what would qualify a good friend for a job. The purpose of this task was to require a similar amount of creativity and cognitive resources as the personal job interview, while not containing the same stressful element of social evaluation and having to "talk oneself up". Finally, all subjects performed their tasks simultaneously with the other participants; this made the individual contributions unintelligible to the passive observer and the other participants, thus further reducing the social evaluative element. One draw-back of subjects talking simultaneously in the control condition is that actual performance during the control task could not be assessed and compared to performance of subjects in the stress groups. Therefore, although the tasks that subjects performed were very similar, we have no comparison of actual cognitive load during the stress and control tasks. Total duration of the task and speaking time for each participant were matched to the parameters of the stress condition, when the speaking time for each part of the control task was finished (3 minutes for the speech and 2 minutes for the arithmetic task), participants were asked to keep a standing position and read neutral magazines for the remaining time.

7.6.3 Intertemporal Choice Task

Titration is a standard method for identifying time preferences in the discounting literature (3-6). The titration worked as follows: for each choice of the later reward, the sooner reward was increased by half the difference between it and 40 CHF; for instance, if a subject chose CHF 40 in 12 months and 1 day over CHF 20 tomorrow, the next trial would offer the subject a choice between CHF 40 in 12 months and 1 day and CHF 30 tomorrow; if the subject still chose CHF 40 in 12 months and 1 day, the next offer would be CHF 40 in 12 months and 1 day vs. CHF 35 tomorrow, and so on. For each choice of the sooner reward, the sooner reward was decreased by half of the difference between it and the previously offered soon reward. For instance, if a subject chose CHF 20 tomorrow over CHF 40 in 12 months and 1 day, the next trial would offer the subject a choice between CHF 10 tomorrow and CHF 40 in 12 months and 1 day; if the subject chose CHF 10 tomorrow, the next offer would be CHF 5 tomorrow vs. CHF 40 in 12 months and 1 day, and so on. The titration procedure lasted for 7 trials at each combination of delays; this means that each indifference point was identified to a precision of CHF 0.156 (CHF 20 x 0.5^7, i.e. the initial difference between CHF 20 and CHF 40/ CHF 0 was halved seven times). The amount of the sooner reward at the end of this titration procedure was taken as the indifference point for the particular delay combination, i.e. the amount of the sooner

smaller reward where participants switched between the smaller sooner and the later larger reward.

This procedure resulted in an individual discount function for each subject, which was used as the basis for fitting parameters of several models of intertemporal choice, described below. Note that this procedure collapses subjects' choices in the time preference task into one or two parameters; thus, each subject entered the statistical analysis only once, i.e. we are not using multiple (non-independent) data points for each subject.

Possible serial correlation and order effects in subjects' responses were controlled for by randomizing the order of trials across blocks, i.e. the order in which the various indifference points were determined. In addition, the side of the screen (left or right) on which the "late" and "soon" options were presented on each trial was randomized across trials.

Note that the soonest option subjects could choose in the intertemporal choice task was "tomorrow". One may ask whether this delay can be considered small enough to be useful in identifying present bias. We chose this design for the following reasons:

First, we found it difficult to include an earlier reward in the design without confounding transaction costs: the chosen option on one of the trials in the intertemporal choice task was paid out for real, i.e., participants could pick up the chosen amount on the chosen day of delivery, using a voucher valid at the University cashier's office. If the smallest delay was "today", choosing this option would result in lower transaction costs compared to choosing a more delayed option, because subjects are already at the University, while at any other delay than today (i.e. "tomorrow", but also in several months) subjects may have to travel to the University specifically to pick up their payment. Therefore, in this case we would have been unable to dissociate transaction costs from present bias.

Second, we did consider other forms of payment than cash vouchers, but they all suffered from similar problems: we judged that getting a check or cash on the day vs. receiving a check or cash in the mail later did not equate the perceived risk of the transaction; bank transfers cannot be effected on the same day and also have to be picked up at the bank before they can be consumed; Amazon vouchers cannot be turned into consumption immediately because of the delays associated with mail orders; mobile phone money transfers and pre-paid debit cards are not available in Switzerland. Thus, the "tomorrow" option seemed to us the cleanest way of eliciting time preference without risk of transaction cost confounds.

Third, note that even with a "today" option, when using money as a reward, it is almost impossible to study true present bias in the sense of the immediate present; at best, one could hope for a time frame of a few hours,

since subjects are unable to spend the money they earn in the experiment until they leave the lab. Thus, even a "today" option would not fully address the disconnect between the "present" and the earliest time at which the reward can actually be consumed.

Fourth, to the extent that we observe present bias in our behavioral data (we consistently find beta < 1), we argue that people actually do consider tomorrow as part of the extended present. Thus, in our view it is likely that, even if it were possible to overcome the difference in transaction costs, similar results would be found if all the payoffs had been one day earlier (i.e. today, in 3 months, in 6 months etc.).

Fifth, note that the delays in our temporal choice task are relatively long, ranging from 3 months to 12 months. In comparison, the delay between today and tomorrow is very small in magnitude, making it likely that tomorrow is in fact interpreted as part of the extended present by our subjects. In support of this claim, comparable studies where the soonest payoff was on the same day find similar discount rates as we do when the soonest payoff is tomorrow (7-9). In particular, estimates of the beta parameter reported in the literature involving, among others, immediate outcomes in real-life or laboratory situations are very similar to those obtained for our control group: our control subjects have beta = 0.82 on average, which is close to the beta = 0.7 reported by Laibson et al. (10), and falls squarely within the "realistic range" of beta from 0.8 – 0.85 argued by Laibson et al. (11).

Finally, note that if "tomorrow" is not considered part of the extended present by our subjects, it is likely that we in fact underestimated the behavioral effect of stress on present bias. Conversely, we would predict that the bi-directional effect of stress on present bias would be stronger if a "today" option was included, since biological theory (see Introduction) suggests that organisms will be less oriented towards the future directly after stress.

7.6.4 Classification into present-biased and non-present-biased subjects

Our experimental design afforded classification of subjects into present-biased and non-present-biased: present-biased subjects are those whose discounting over a given period is greater when that period is in the near future compared to when it is in the more distant future. In our design, a present-biased subject would discount more between tomorrow and 3 months and 1 day than between 3 months and 1 day and 6 months and 1 day; similarly, they would discount more between tomorrow and 6 months and 1 day than between 6 months and 1 day and 12 months and 1 day. We can use this feature of

our experimental design to directly classify subjects into present-biased and non-present-biased, without assumptions about the shape of the discount function. We classified a subject as present-biased as follows: we obtained the difference between their "tomorrow vs. 3 months and 1 days" indifference point and the "3 months and 1 day vs. 6 months and 1 day" indifference points from the intertemporal choice task; similarly, we computed the difference between their "tomorrow vs. 6 months and 1 days" indifference point and the "3 months and 1 day vs. 12 months and 1 day" indifference points. A subject was classified as present-biased when the average of these two differences was greater than zero, i.e. when on average they discounted more over periods of 3 to 6 months when those periods were in the near future (beginning tomorrow) compared to the distant future (beginning in 6 months and 1 day). The resulting classification is a dummy variable that is 1 when a subject is present-biased based on this definition, and zero otherwise.

7.6.5 Model Fits

7.6.5.1 Hyperbolic discounting model

In addition to the beta-delta model, we also fitted a standard hyperbolic model (12-16) of the following shape:

$$V_t = \frac{A}{1+kt},$$

where V_t indicates the discounted value at time t, A is the amount of reward, t the delay until reward delivery, and k is a single parameter describing the shape of the hyperbola. Because we expressed the value of the delayed rewards as a fraction of the value of the immediate reward, $A = 1$.

7.6.5.2 Exponential discounting model

Finally, we fit a standard exponential discount function of the following shape:

$$V_t = A \exp\left(-\delta^t\right),$$

where V_t indicates the discounted value at time t, A is the amount of reward, t the delay until reward delivery, and δ is the parameter describing the steepness of the exponential discount function. Because we expressed the value of the delayed rewards as a fraction of the value of the immediate reward, $A = 1$.

7.6.6 Statistical Analyses

7.6.6.1 Definition of cortisol responders

Cortisol responders were defined by an absolute cortisol increase of at least 2.5 nmol/l (1, 17) in response to the stressor (t5-t2). This resulted in 16 of 20 cortisol responders in the early stress group and 17 of 20 responders in the late stress group. Stress-induced increases in salivary cortisol and alpha-amylase (sAA) to probe stress reactivity were calculated with a formula for area-under-the-curve increases (AUCi) from baseline (18).

7.6.6.2 Performance on the intertemporal choice task

For our main analysis, performance on the intertemporal choice task was analyzed by testing the model parameters with a 2 (Stress: TSST-G vs. Control) x 2 (Timing: Early vs. Late) MANOVA with age and body mass index (BMI) as covariates. Cortisol is known to be influenced by age and BMI (19-25); for this reason it is standard procedure to include these variables as covariates in statistical analyses in stress research (based on (26); e.g. (27).

In regression terms, this analysis can be written as

$$beta_i = \beta_0 + \beta_1 stress_i + \beta_2 delay_i + \beta_3 stress_i \cdot delay_i + \beta_4 \mathbf{X_i} + u_i$$

$$delta_i = \beta_0 + \beta_1 stress_i + \beta_2 delay_i + \beta_3 stress_i \cdot delay_i + \beta_4 \mathbf{X_i} + u_i,$$

where $beta_i$ and $delta_i$ are the present bias and impatience parameters, respectively, in Laibson's discounting model, $stress_i$ is a dummy variable indicating whether subject i was in the stress group or the control group, $delay_i$ is a dummy variable indicating whether subject i was tested immediately after stress (early) or 20 minutes later (late), $\mathbf{X}_i$ is a vector of covariates including age and body mass index, and u_i is the error term.

7.6.6.3 Classification into present-biased and non-present-biased subjects

For a more direct test of whether stress increased present bias, we asked whether the number of subjects classified as present-biased based on the dummy variable described above was affected by stress and delay. To this end, we performed a 2 (Stress: TSST-G vs. Control) x 2 (Timing: Early vs. Late) ANOVA with the present bias dummy as the dependent variable and age and body mass index (BMI) as covariates. We report results both when cortisol responders are excluded from the analysis and when they are included. In regression terms, this analysis can be written as

$$presentbiased_i = \beta_0 + \beta_1 stress_i + \beta_2 delay_i + \beta_3 stress_i \cdot delay_i + \beta_4 \mathbf{X_i} + u_i,$$

where $presentbiased_i$ is the dummy variable classifying subjects into present-biased and non-present-biased, and the other variables are as described above. Results without covariates are reported in the Supplementary Results section.

As in the main analysis, in case of significant main and interaction effects, planned comparisons were performed, for which one-tailed tests are justified; however, we report the results for two-tailed tests as well.

7.6.6.4 Exclusion of non-responders and selection bias

To assess whether the exclusion of non-responders led to selection bias, we regressed the probability of being a non-responder on a number of observable variables. Specifically, we fit the following model using OLS:

$$nonresponder_i = \beta_0 + \beta_1 age_i + \beta_2 bmi_i + \beta_3 baselinestress_i$$

$$+\beta_4 baselinecort_i + \beta_5 beta_i + \beta_6 delta_i + \beta_7 barratt_i + u_i,$$

where $nonresponder_i$ is a dummy for being a cortisol non-responder to stress (for subjects in the stress condition), $baselinestress_i$ is self-reported stress before the stress induction procedure on a scale from 0 (not at all stressed) to 100 (very stressed), $baselinecort_i$ is baseline cortisol in nmol/l, $beta_i$ and $delta_i$ are the present bias and impatience parameters, respectively, in Laibson's discounting model, and $barratt_i$ is trait impulsivity as measured by the Barratt scale. u_i is the error term.

7.6.6.5 Relationship between hormone increases and stress-induced changes in discount parameters

The relationship between hormone increases and stress-induced changes in discount parameters was assessed by OLS regression of model parameters on area-under-the-curve for cortisol and sAA using heteroskedasticity-robust standard errors. Specifically, separately for the early and late stress groups, we fit the following models:

$$beta_i = \beta_0 + \beta_1 cortAUC_i + u_i$$

$$delta_i = \beta_0 + \beta_1 cortAUC_i + u_i,$$

where $cortAUC_i$ is cortisol area-under-the-curve, and $beta_i$ and $delta_i$ are the present bias and impatience parameters, respectively, in Laibson's discounting model. u_i is the error term.

7.7 Supplementary Results

7.7.1 Classification into present-biased and non-present-biased subjects

Our experimental design afforded classification of subjects into present-biased and non-present biased without assumptions about the shape of their discount function: a subject was classified as present-biased when their discounting over a given period was larger on average when that period was in the near future compared to when it was in the more distant future (see SI Materials and Methods). In line with the findings reported above, stress differentially affected the number of subjects who were classified as present biased: in the early testing group, 15 out of 20 subjects were classified as present-biased under control conditions, while 19 out of 20 subjects who had undergone the TSST-G were classified such. In the late testing group, 20 out of 21 control subjects, but only 15 out of 20 TSST-G subjects, were present-biased. These differences were statistically significant: we observed a two-way stress x time interaction on the present bias dummy variable ($F_{1,68}$ = 7.82, P = 0.007 (two-tailed)). Planned comparisons revealed that when tested immediately after stress, stressed subjects were more likely to be classified as present biased than controls ($F_{1,32}$ = 6.43, P = 0.008 (one-tailed) / P = 0.016 (two-tailed)). By contrast, stressed individuals tested at the later time-point were less likely to be classified as present-biased than controls ($F_{1,34}$ = 3.48, P = 0.036 (one-tailed) / P = 0.071 (two-tailed)). These effects remained significant when cortisol non-responders were included in the analysis (stress x delay interaction: $F_{1,74}$ = 5.76, P = 0.019 (two-tailed); early stress vs. control: $F_{1,36}$=3.90, P = 0.028 (one-tailed); late stress vs. control: $F_{1,36}$=3.14, P = 0.043 (one-tailed)), and when no covariates were included in the model (stress x delay interaction: $F_{1,70}$ = 7.89, P = 0.006 (two-tailed); early stress vs. control: $F_{1,34}$ = 5.04, P = 0.015 (one-tailed); late stress vs. control: $F_{1,36}$ = 2.97, P = 0.046 (one-tailed)). Note that this analysis is only based on whether a subject is present biased or not. Conclusions about the degree of present bias, and differences in the degree between stress and control group are not addressed here, but reported in the main article.

Stress and intertemporal choice performance including non-responders. To ascertain that the exclusion of non-responders does not lead to selection

bias, we included non-responders in the data pool and re-ran the analysis: We replicated the interaction effect between stress and timing on present bias (beta parameter in the beta-delta model) in the intertemporal choice task when all subjects (also non-responders) were included (stress x time interaction: $F_{1,74} = 4.07$, $P = 0.047$ (two-tailed); early stress vs. control: $F_{1,36} = 1.22$, $P = 0.27$ (two-tailed)/ $P = 0.14$ (one-tailed); late: $F_{1,36} = 4.34$, $P = 0.044$ (two-tailed)/ $P = 0.022$ (one-tailed)).

7.7.2 Exclusion of non-responders and selection bias

To corroborate on the finding that the exclusion of non-responders does not lead to selection bias, we regressed the probability of being a cortisol non-responder to stress (see SI Materials and Methods for definition) on a number of observable variables, including age, body mass index (BMI), baseline cortisol levels, baseline self-reported stress on the visual-analog scale, Laibson's beta and delta parameters, and trait impulsivity as measured by the Barratt scale. None of these variables successfully predicted whether a participant was a responder or a non-responder (OLS coefficients and P-values: age, coeff = -0.033, P = 0.270; bmi, coeff = 0.013, P = 0.607; baseline self-reported stress, coeff = -0.002, P = 0.689; baseline cortisol, coeff = 0.007, P = 0.397; beta, coeff = 0.347, P = 0.330; delta, coeff = -0.583, P = 0.554; barratt, coeff = -0.099, P = 0.153). Thus, non-responders are similar to responders on important variables, and their exclusion is unlikely to have introduced significant selection bias.

Model Fits. Our data were better described by Laibson's quasi-hyperbolic model than the standard hyperbolic discounting model or the standard exponential model (Akaike Information Criteria: Laibson model, -11.80; hyperbolic model, -9.22; exponential model, -8.46). Nevertheless, to support our findings, we also fit the standard hyperbolic model (12)) and examine the effect of stress on the model's k parameter. The results were similar to those obtained for the beta parameter in Laibson's quasi-hyperbolic model (excluding non-responders: stress x time interaction: $F_{1,68} = 4.46$, $P = 0.038$ (two-tailed); early stress vs. control: $F_{1,32} = 2.72$, $P = 0.055$ (one-tailed); late: $F_{1,34} = 2.18$, $P = 0.075$ (one-tailed); including non-responders: stress x time interaction: $F_{1,74} = 3.24$, $P = 0.076$ (two-tailed); early stress vs. control: $F_{1,36} = 2.23$, $P = 0.072$ (one-tailed); late stress vs. control: $F_{1,36} = 1.12$, $P = 0.148$ (one-tailed)). Similarly, the results from the standard exponential model were similar to those obtained with Laibson's quasi-hyperbolic model (excluding non-responders: stress x time interaction: $F_{1,68} = 4.32$, $P = .042$ (two-tailed); early stress vs. control: $F_{1,32} = 2.41$, $P = 0.066$ (one-tailed); late stress vs. control: $F_{1,34} = 2.03$, $P = 0.082$ (one-tailed); including non-

responders: stress x time interaction: $F_{1,74} = 3.47$, $P = 0.066$ (two-tailed); early stress vs. control: $F_{1,36} = 2.31$, $P = 0.069$ (one-tailed); late stress vs. control: $F_{1,36} = 1.18$, $P = 0.142$ (one-tailed)). Note that these results reach lower levels of significance than those from the quasi-hyperbolic model; this is expected, as our data is best described by the quasi-hyperbolic model (see information criteria above), and the functional forms imposed by the standard exponential model and the standard hyperbolic model may not adequately account for the structure of the data. In particular, these models conflate present bias and impatience; however, as shown above, this distinction is necessary because the behavioral effect of stress on discounting appears to be driven by present bias and not impatience.

7.7.3 Inconsistency of responses in the time preference task

A possible confound to our results is that subjects' responses to the time preference questions may be inconsistent, and that this inconsistency may be affected by stress. This is an important concern, since it has been shown that e.g. the effect of working memory load on discounting can be explained by increased randomness in responses under higher working memory load, rather than truly different time preferences (28, 29). In principle, this mechanism could underlie our results as well.

However, this possibility is unlikely for the following two reasons. First, note that inconsistency would have to be affected by stress in two different directions to account for our results: in the early condition, stress would have to increase inconsistent responding, while in the late condition, stress would have to decrease inconsistent responding. We are not aware of theory or evidence that would predict such a bidirectional effect.

Second, we can directly assess whether our subjects showed inconsistent responses in the time preference task, and whether these responses were affected by stress. To answer this question, we analyzed whether our subjects showed non-monotonic discount functions, i.e. whether any indifference point identified by our titration algorithm at a particular delay was lower than any indifference point at a later delay. Note that our experimental design did not make it possible for subjects to give inconsistent responses when identifying any particular indifference point, since the indifference points were approximated by a titration algorithm. Thus, any particular indifference point, by the nature of the algorithm, was obtained through choices that were "consistent" by definition. However, mistakes that subject made during the titration would manifest themselves in inconsistent indifference points;

this is what we analyze in the following.

Overall, 24% of indifference points showed evidence of inconsistency. However, the proportion of inconsistent responses for a particular subject was not dependent on whether this subject was in the stress or control conditions; in an OLS regression with the percentage of inconsistent indifference points as the dependent variable, and stress and delay, and their interaction, as the independent variables, no coefficient was significant (stress: t=0.94, p=0.35; delay: t=-0.32, p=0.75; stress x delay: t=-0.91, p=0.37). Thus, the effect of stress on present bias that we observe is not due to differential likelihood of inconsistent responses across the stress and control conditions.

7.7.4 Partial cortisol correlations and mood regulation account

A further potential concern is that the responses in the intertemporal choice task themselves may have influenced cortisol levels; in particular, in the early group, subjects may have made particular choices to regulate their own mood after stress induction. However, note that we observe the opposite: subjects in the early group who are particularly likely to choose immediate rewards in fact show higher overall cortisol levels, rather than lower levels. This is even true for cortisol levels after the intertemporal choice task (from 0 to 50 minutes after the TSST-G or control task): subjects with particularly low betas (i.e. strong present-bias) in fact show somewhat higher cortisol levels even during this period; a regression of cortisol area-under-the-curve for only the time period after the behavioral task on beta yields a non-significant but negative coefficient on beta (coeff: -987.3, P = 0.364). This means that cortisol levels after the behavioral task cannot be predicted by choices in the behavioral task, and if anything, this effect goes in the opposite direction as the one we would expect based on a mood regulation account. Conversely, the correlation between stress-induced cortisol increases and present bias is also observed in the early group when considering only cortisol measurements until the time of the intertemporal choice task for calculating area under the curve, although this result has poor statistical power due to the reduction in the number of measurements per subject (regression of beta on cortisol area under the curve, coeff = -0.001, P = 0.114). Note that behavioral testing of the late group occurred immediately before the end of the experiment, thus precluding that observed cortisol levels are biased by task performance in this group. In the late group, no correlation between stress-induced cortisol increase and present bias is observed when considering only timepoints until just after the stress task (coeff = 4 x 10^{-5}, P = 0.256); this supports our con-

jecture that late-developing effects of cortisol are responsible for the reversed finding in this group compared to the early group.

A further test of whether mood regulation might account for our results is to attempt to predict the time preference parameters in the stress group not with stress-induced cortisol increases, but with stress-induced changes in negative affect and subjective stress (we thank a reviewer for this suggestion). A relationship between the change in these variables due to the stress task and the change in time preference parameters that goes in the same direction as the cortisol effect would suggest that changes in affect could underlie this effect. However, if anything we observe the opposite: a greater response to the stress task in terms of negative affect or subjective stress is not significantly associated with present bias; there is a trend, but it goes in the opposite direction as the cortisol effect: a larger response to the TSST-G in terms of negative affect or subjective stress is non-significantly associated with larger beta parameters in the early group (OLS regression of beta on the after-before difference in negative affect: coeff = 0.026, P = 0.080; subjective stress after-before: coeff = 0.004, P = 0.111), and smaller beta parameters in the late group (negative affect: coeff = -0.007, P = 0.088; subjective stress: coeff = -0.002; P = 0.255). Thus, the mood changes induced by the stress task have an effect on the discount parameters that counterveils the relationship between cortisol and present bias in the stress group, and also runs against the effect of stress across stress and control groups. Mood regulation is therefore unlikely to be the underlying driver of our results.

7.7.5 Cortisol profiles in the early and late groups

A further test of whether choices in the time preference task might have affected cortisol levels is to compare the cortisol profiles of the early and late groups separately for both the stressed and control subjects: if the intertemporal choice task affected cortisol levels, then cortisol levels should be different in the early vs. late groups, since the early group performed the behavioral task early enough that it might be reflected in cortisol levels, while the late group did not. The ANOVAs showed that interactions between Sample Period and Delay were not significant for subjects in either the control ($F_{1.9,73.4}$ = 0.102, P = 0.893) or the stress groups ($F_{1.4,54.7}$ = 0.781, P = 0.425). Furthermore, for both the stress and control groups none of the planned simple contrasts related to baseline were significant (all Ps > 0.10). In addition, cortisol AUC levels did not significantly differ between the early and late subjects within both the control (t_{39} = -0.095, P = 0.925) and the stress groups (t_{38} = -0.853, P = 0.399). Thus, we did not find any evidence that cortisol levels differ between the early and late groups, ren-

dering it unlikely that choices in the time preference task affected cortisol levels.

Bibliography

[1] Kirschbaum C, Pirke KM, & Hellhammer DH (1993) Neuropsychobiology 28, 76-81.

[2] von Dawans B, Kirschbaum C, & Heinrichs M (2011) Psychoneuroendocrinology 36, 514-522.

[3] Mazur JE (1988) J Exp Anal Behav 49, 37-47.

[4] Green L & Myerson J (2004) Psychol Bull 130, 769-792.

[5] Kable JW & Glimcher PW (2007) Nat Neurosci 10, 1625-1633.

[6] Rachlin H, Raineri A, & Cross D (1991) J Exp Anal Behav 55, 233-244.

[7] Benzion U, Rapoport A, & Yagil J (1989) Management Science 35.

[8] Thaler RH (1981) Economic Letters 8, 201-207.

[9] Frederick S, Loewenstein G, & O'Donoghue T (2002) Journal of Economic Literature 40, 351-401.

[10] Laibson D, Repetto A, & Tobacman J (2007) National Bureau of Economic Research Working Paper No. 13314.

[11] Laibson D, Repetto A, & Tobacman J (1998) Brookings Papers on Economic Activity 1, 91-196.

[12] Mazur JE (1984) Journal of Experimental Psychology: Animal Behavior Processes 10, 426-436.

[13] Chung SH & Herrnstein RJ (1967) J Exp Anal Behav 10, 67-74.

[14] Kirby KN & Herrnstein RJ (1995) Psychological Science 6, 83-89.

[15] Ainslie G (1975) Psychol Bull 82, 463-496.

[16] Green L & Myerson J (1996) American Zoologist 36, 496-505.

[17] Kirschbaum C, Wolf OT, May M, Wippich W, & Hellhammer DH (1996) Life Sci 58, 1475-1483.

[18] Pruessner JC, Kirschbaum C, Meinlschmid G, & Hellhammer DH (2003) Psychoneuroendocrinology 28, 916-931.

[19] Larsson CA, Gullberg B, Rastam L, & Lindblad U (2009) BMC Endocr Disord 9, 16.

[20] Ukkola O, Gagnon J, Rankinen T, Thompson PA, Hong Y, Leon AS, Rao DC, Skinner JS, Wilmore JH, & Bouchard C (2001) Eur J Endocrinol 145, 1-9.

[21] Travison TG, O'Donnell AB, Araujo AB, Matsumoto AM, & McKinlay JB (2007) Clin Endocrinol (Oxf) 67, 71-77.

[22] Purnell JQ, Brandon DD, Isabelle LM, Loriaux DL, & Samuels MH (2004) J Clin Endocrinol Metab 89, 281-287.

[23] Strahler J, Mueller A, Rosenloecher F, Kirschbaum C, & Rohleder N (2010) Psychophysiology 47, 587-595.

[24] Keenan DM, Roelfsema F, Carroll BJ, Iranmanesh A, & Veldhuis JD (2009) Am J Physiol Regul Integr Comp Physiol 297, R515-523.

[25] Veldhuis JD, Iranmanesh A, Roelfsema F, Aoun P, Takahashi P, Miles JM, & Keenan DM (2011) J Clin Endocrinol Metab 96, 2874-2881.

[26] Wardle J, Chida Y, Gibson EL, Whitaker KL, & Steptoe A (2011) Obesity (Silver Spring) 19, 771-778.

[27] Bostock S, Hamer M, Wawrzyniak AJ, Mitchell ES, & Steptoe A (2011) Psychoneuroendocrinology 36, 1175-1183.

[28] Hinson JM, Jameson TL, & Whitney P (2003) J Exp Psychol Learn Mem Cogn 29, 298-306.

[29] Franco-Watkins AM, Pashler H, & Rickard TC (2006) J Exp Psychol Learn Mem Cogn 32, 443-447; discussion 448-450.

Part II

Other Work

Chapter 8

The Social Costs of Randomization

8.1 Summary

Randomized allocation of treatment to participants is the gold standard for identifying the welfare effects of medical and social interventions. Here we show that this allocation mechanism has social costs, in the sense that participants, in particular those in the control group, perceive the random allocation mechanism as unfair and incur costs to signal this. Second, we show that the perceptions of fairness differ systematically between those who receive treatment and those who decide about its allocation; specifically, recipients perceive randomization as uniformly unfair, whereas allocators judge it as fair when the relative wealth of recipients is similar. These findings have implications for the value of targeting: investing resources into finding deserving participants is justified because not doing so generates social costs in terms of perceived unfairness of random allocation.

8.2 Introduction

The core question of economics is: how are scarce resources distributed (Robbins, 1932)? Throughout history, humans have made decisions about the allocation of resources not only for themselves, but also on behalf of others: parents distribute food among their children, rulers allocate unemployment benefits and healthcare among their citizens, NGOs distribute aid among people in developing countries. But according to what criteria should scarce resources be distributed among potential recipients? This question lies at the heart of Social Choice Theory, and has been the subject of intense de-

bate for centuries (Rawls, 1971; Sen, 1970; Bentham, 1789; Arrow, 1951; Harsanyi, 1955). Most social choice theory today operates within a utilitarian framework. At a minimum, this means that the utility that each individual gains from being given a particular resource should be considered in the allocation decision: the blind don't benefit much from being given spectacles, the hungry may benefit more from food than the satiated, and to the utilitarian, these facts should inform the decision about who gets what. However, considering the utility of the affected individuals also makes social choice in this framework exceedingly difficult, since it requires making interpersonal comparisons of utility (ICU); Robbins (1932) famously argued that such comparisons are almost impossible in principle. In the extreme case, when no information is available about the mental states or material resources of potential recipients, how should allocation proceed?

The most common answer to this question is randomization: when no detailed information is available about potential recipients, it is standardly argued that randomization should be the method of choice (Eckhoff, 1989; Katta & Sthuraman, 2006). The casting of lots is an ancient and well-trodden path to achieve fair allocations; its history goes back at least to ancient Greece, where membership in the Athenian Council of 500 in the 4th and 5th centuries B.C. was determined by drawing lots among all qualified persons (Eckhoff, 1989). United States juries are still chosen through randomization today. In an 1842 case of a sinking boat in which passengers had to be thrown overboard to ease the load on the leaking vessel, the crew decided to throw single men overboard and spare married men, children, and women; a court later held that this decision mechanism was unjust, and that lots should have been drawn instead (US vs. Holmes, 1842). Randomization is also widely used in allocating land rights: an instruction to divide land ownership by lot in inheritances is found in the Book of Numbers (Silverman & Chalmers, 2001); similarly, the Vikings divided land among heirs or other claimants by lot, and a law to this effect still exists today in Denmark for cases where the estate of a deceased person is administered by a court (Eckhoff, 1989). Randomization is also frequently invoked in military drafts: examples include Britain in the 18th century before an impending French invasion of Ireland, Austria-Hungary and the United States before World War I, and famously, during the Vietnam War (Silverman & Chalmers, 2001; MacAtasney, 1998; Fienberg, 1971; Rosenblatt & Filliben, 1971). (Note, however, that randomization in these cases is not undisputed; for instance, economists have argued that the coercion and cost associated with this system should be replaced by one in which the military competes for volunteers in the labor market; Fisher, 1969.) Other examples include lotteries held for dorm roommates at colleges and the U.S. green card lottery.

Thus, randomization is of central importance in social choice theory: it is the single method that is claimed to be fair and efficient when no information is available about the mental states or material resources of potential recipients (Eckhoff, 1989; Katta & Sethuraman, 2006). In addition, however, randomization has another crucial function in science and medicine: it is the backbone of the Randomized Controlled Trial (RCT). In randomized controlled trials (RCTs), the randomization mechanism ensures that the treatment and control groups are statistically identical, and thus allows attributing any differences in behavior or welfare outcomes to the intervention. It is due to this feature that RCTs are widely regarded as the mainstay of clinical trials (Sacks et al., 1981) and, more recently, the evaluation of social programs in both developed and developing countries (Fisher, 1928; Newman et al., 1994; Harrison & List, 2004; Duflo & Kremer, 2003; Thomas, 2010). Like randomized allocation of resources in general, randomization in the service of science has a long and proud history. The Belgian physician van Helmont first suggested randomly assigning patients to a bloodletting treatment and a control treatment without bloodletting, and then ask "how many funerals both of us shall have" (van Helmont, 1662). Systematic randomized trials appeared by the 19th century (Chalmers, 2001).

The criterion commonly required for the permissibility of randomization in the context of clinical trials and field experiments is clinical equipoise. This principle argues that for a randomized controlled trial to be justified, clinicians must be genuinely uncertain as to which of several treatments is better (Freedman, 1987). However, in the case of many welfare programs, equipoise does not obtain: it is difficult to argue genuine uncertainty as to whether receiving a welfare benefit is better or worse than not receiving it. The same will apply in the context of the experiment described in this paper, where one participants receives money and the other does not; in this case it is difficult to argue that not receiving money is as good as receiving money. However, a number of authors have argued that lack of equipoise is not necessarily sufficient grounds for rejecting randomization as an allocation method; for instance, randomization is permissible if resources are scarce and not everyone can get the benefit – in this case randomization may even be the preferred allocation method (Lockwood & Anscombe, 1983; Lilford & Jackson, 1995; Toroyan et al., 2000).

Thus, social choice theorists have generally argued that randomization is acceptable in social choice when no information is available about the feature of potential recipients; and in the context of RCTs, when resources are so scarce that only some potential recipients can receive the benefit. However, is this actually true empirically – i.e., do people actually view randomization as fair under these conditions? This question is important in its own right

– if randomization is not actually perceived as fair by potential recipients, this would place a serious constraint on its permissibility. Second, in the context of RCTs, participants' perception of the fairness of the allocation mechanism may actually influence their behavior: it has been shown that the mere process of being surveyed alters respondents' behavior (Zwane et al., 2011; Orne, 1962; Bhargava, 2006), and thus it is possible that behavior may also respond to the allocation of treatment or control through randomization.

The perceived fairness of randomization has only been considered in a handful of studies, all of them survey-based. Hillis & Wortman (1976) found in a survey that randomization was perceived to be permissible when the study was scientifically necessary; however, these authors also found that scarce resources were not regarded as a sufficient justification for randomization. Innes (1979) presented college students with vignettes about a research project and then asked them for their assessment of the justifiability of using randomization in the study. Randomization was judged positively throughout. Erez (1985) surveyed prison inmates about their opinions regarding four different selection criteria for special programs from which they might benefit: need; merit; first come, first served; and random assignment. Need was perceived as the fairest and randomization as the least fair criterion. Similarly, Johnson found that people generally judge randomization as unacceptable in clinical trials when one treatment is better than the other: even if expert opinion is split 80%-20% about which treatment is better, only 3% of respondents find randomization acceptable. However, acceptability was better when the treatment was not a life-saving intervention. This finding echoes Gary Burtless' (1995) claim that "except among philosophers and research scientists, random assignment is often thought to be an unethical way to ration public resources."

But is it? Surprisingly, no study to date has gone beyond using surveys and tested the perceived fairness of randomization in an experimental setting. The purpose of the present study is to fill this gap. We set up experimental groups of three participants, in which one allocator decides on how to allocate an indivisible prize of 5 CHF to one of the other two receivers. The allocator can choose between one of the two receivers herself, or she can choose to let the computer randomize. We then ask whether the receivers judge the different allocation strategies as fair; crucially, we elicit this information in an incentive-compatible manner, in that we test whether receivers are willing to incur costs to reward or punish the allocator for her decision. In addition, we manipulate the information the allocator has about the receivers: they can either have the same or a different level of wealth, and this is known either with complete certainty or with some uncertainty.

We find that randomization is generally not perceived as fair: receivers

punish the allocator when she chooses random allocation. In addition, this punishment is not sensitive to equality or uncertainty: the two receivers do not punish the allocator less when they have equal incomes, or when their incomes are not known with certainty. In contrast, the allocator responds to information about the receivers: when their wealth is similar, allocators are more likely to randomize allocation. Together, our results suggest that random allocation of resources has social costs: receivers are willing to incur costs to signal their discontent with randomization as an allocation mechanism. Second, our findings reveal an important disconnect between what allocators and receivers perceive as fair: allocators deem it acceptable to randomize when incomes are similar, whereas this is not true for receivers. These findings have implications for the design of allocation mechanisms in social choice.

8.3 Materials & Methods

8.3.1 Participants

We tested 105 healthy participants who were recruited from the subject pool at the University of Zürich. Their mean age was 22.08 ± 3.31 (mean ± S.D.). We excluded students of economics and psychology. All participants gave written informed consent and were reimbursed for their participation. An experimental session lasted 2h.

8.3.2 Session structure

The experiment was conducted in three sessions with 36, 36, and 33 participants, respectively. Participants were seated at networked computers in the behavioral laboratory of the Department of Economics at the University of Zürich. Each participant was randomly assigned the role of allocator or receiver at the beginning of the session, and kept this role for the entire session. Participants played the task in groups of 3, where two participants were receivers, and the remaining participant was the allocator. All participants knew of their role (allocator vs. receiver), but did not know the identities of the other players in their group. Allocators received a starting endowment of CHF 32; receivers received varying starting incomes, depending on the condition (See details below).

8.3.3 Block structure and conditions

After being given detailed task instructions and correctly answering test questions about the task, participants performed N/3 blocks of the allocation task; thus, in the sessions with 36 participants, 12 blocks were played, and in the session with 33 participants, 11 blocks. Each block consisted of 6 decision situations, each corresponding to one of 6 conditions. In the "richer-certain" condition, receiver A had a higher starting endowment than receiver B. In half of the blocks, this starting endowment was CHF 20, while that of receiver B was CHF 10; in the other half of blocks, receiver A's endowment in this condition was CHF 30, while that of receiver B was CHF 20. In the "poorer-certain" condition, these roles were reversed: receiver A either had an endowment of CHF 20 while receiver B had CHF 10, or receiver A had CHF 30 while receiver B had CHF 20. In the "equal-certain" condition, both receivers had the same starting income; in half of the blocks, both receivers had CHF 15, in the other half of blocks, they both had CHF 25. Thus, the mean endowments of the "richer-certain" and "poorer-certain" conditions across participants were equal to the endowment in the "equal-certain" condition.

These three conditions were replicated with the addition of uncertainty about incomes to generate the remaining three conditions: in the "richer-uncertain" condition, either receiver A had a starting endowment somewhere between CHF 12.50 - CHF 27.50 while receiver B had an endowment between CHF 2.50 - CHF 17.50; or receiver A had an endowment somewhere between CHF 22.50 - CHF 37.50 while receiver B had an endowment between CHF 12.50 - CHF 27.50. In the "poorer-uncertain" condition, these roles were again reversed, as described above. In the "equal-uncertain" condition, either both participants had an endowment between CHF 7.50 - CHF 22.50, or between CHF 17.50 - CHF 32.50. Within these ranges, a uniform probability distribution was used to determine the actual starting endowments. receivers were not informed about the probability distribution of their incomes within the ranges; note, however, that the midpoint of the ranges correspond to the endowments in the "certain" conditions. Note also that the endowment ranges of both participants overlapped in the uncertain conditions; thus, in the "richer-uncertain" condition, participant A had a higher endowment participant B in expectation.

Within each block, participants played one trial in each of these conditions; thus, against each allocator and each other receiver, receivers assumed the "richer-certain", "poorer-certain", "equal-certain", "richer-uncertain", "poorer-uncertain", "equal-uncertain" roles exactly once.

8.3.4 Trial structure

Each trial was structured as follows. At the beginning of the trial, all three members of a group were informed about the endowments of the two receipients; thus, each receiver knew both their own and the other receiver's endowment, and the allocator knew both receivers' endowments. In addition, all members knew that they all had full information; in particular, receivers were aware that the allocator knew their respective endowments.

The allocator then faced the following choice: they could decide how to allocate an indivisible prize of CHF 5 between the two receivers. In doing so, they had three options: they could either give the prize to receiver A, or to receiver B, or they could let the computer randomize with 50-50 probability which receiver would get the prize.

Simultaneously, receivers A and B had the option to punish or reward the allocator for their decision. In particular, each receiver was given an additional reward/punishment budget of CHF 8, and could decide to spend between 0-8 CHF on rewarding or punishing the allocator. (Note that we used neutral language, rather than the terms "reward" and "punishment", in communicating this part of the experiment to the participants; in particular, participants were told that they could "increase or decrease the income of the allocator".) Crucially, reward and punishment could be made contingent on the possible allcoator decisions: for instance, receivers could reward or punish the allcoator for giving the prize to them, to the other receiver, or for choosing to let the computer randomize. We used the strategy method to avoid censoring of data; thus, receivers chose a reward or punishment for each possible allocator decision. Allocators knew that receivers had this reward/punishment opportunity. The reward/punishment technology was 1:2, i.e. for each CHF that receivers spent for rewarding the allocator, CHF 2 were added to the allocator's income; for each CHF that receivers spent to punish the allocator, CHF 2 were subtracted.

After each group of 3 (2 receivers and 1 allocator) had played 6 trials, corresponding to the 6 conditions, groups were randomly re-assigned, and the next block began. In reassigning groups, no participant ever played with any of the other two participants in their group again during the remainder of the experiment. In addition, neither the allocator nor the receivers were informed of the decisions of the other group members at the end of a trial; rather, this information was only revealed at the very end of the experiment. Together, the reassignment without replacement and lack of information about the behavior of others rules out reputational effects, as well as learning and updating about the behavior of others.

8.3.5 Estimates of others' preferences

To control for participants' beliefs about the preferences and actions of others, in the second part of the session, all participants repeated the same task as above, except they were now asked to indicate not their own preferences, but their best estimates of the choices the *other* participants made in each situation. Thus, participants were presented with the same decision situations as in the first part of the experiment, and contingent on their - now fictive - endowment and the various potential allocator decisions, were asked to guess the average reward and punishment that the other participants had dealt the allocator for each income situation and allocator decision. Allocators also guessed the behavior of receivers in this part of the experiment. This task was incentivized by paying each participant CHF 1 for each guess, minus CHF 0.10 for every CHF they deviated from the actual mean that was spent by the receivers in the first part of the experiment to reward or punish the allocator in the particular situation.

8.3.6 Payment

At the end of the experiment, one trial from each part of the experiment was chosen at random and paid out to all participants; thus, the allocator received their initial endowment of CHF 32 plus or minus the aggregate reward or punishment from the receivers for the allocator's decision on this trial; conversely, the receivers received their initial endowment plus the CHF 8 reward/punishment budget, minus the money spent out of this budget on rewarding or punishing the allocator; one of the receivers in addition received the CHF 5 prize, either because of the allocator's decision or through randomization. In addition, all participants received the payment from the second part of the experiment, as described above.

At the end of the experiment, all participants filled out a socioeconomic questionnaire and were paid out.

8.4 Results

Our experiment offers three unique angles to assess participants preferences over appropriate allocations. Firstly, we observe how allocators preferences inform their allocation of scarce goods. Secondly, we observe how receivers choose to reward or punish allocators for their choices and, thirdly, we observe what participants believe about other peoples preferences over allocation decisions.

8.4.1 Allocators' preferences

We begin by presenting simple summary statistics on allocator behavior in Table 8.4.1. The table shows the frequency of allocators actions (whether to give to the richer receiver, the poorer receiver or choose to allocate randomly) under various informational conditions. Overall, allocators favor giving to poor receivers, which they do in 47% of cases. This is followed by allocating randomly, in 35% of cases. Seldom (in 7% of cases) do allocators choose to award the payment to the richer receiver (Note these percentages do not sum to 100 as there were some cases where wealth was equal and choosing the "richer" or "poorer" receiver was not possible.) When we varied whether the allocator knew for certain who was rich and who was poor or whether they knew so only in expectation, we see a shift towards favoring random allocation. In Table 8.4.2 we assess whether this shift is statistically signicant by regressing 3 separate indicator variables (one for each type of allocator choice) on an indicator variable that there was uncertainty about which receiver was richer. The results show that allocators are 13% more likely to randomize and 9% less likely to give to the poorer (in expectation) receiver when there is uncertainty about which receiver is richer.

We further note that when wealth was equal among receivers, randomization was, on average, the preferred method of allocation, but we note that a sizable fraction of allocators (slightly over 30%) chose not to allocate randomly when receivers had equal wealth. Table 8.4.3 shows the frequency with which allocators chose to randomize in various information conditions. We present an F-test that the frequency is equal to 50% - showing that randomization is the preferred option when wealth is equal (with certainty or in expectation) and the less preferred option when wealth is unequal (with certainty or in expectation).

8.4.2 Receivers' preferences

Receivers had a chance to express their preferences over the decisions of allocators by indicating how much they would reward or punish allocators for certain actions. In Table 8.4.4 we show the average reward (or punishment if less than zero) in Swiss Francs under various wealth conditions and in response to different actions on behalf of the Allocator.

The first column, indicating the average reward or punishment a receiver assigns to the allocator when the allocator awarded the payment to that receiver shows an almost uniform reward to the allocator from the receiver who gets the payment - it does not vary much depending on whether the receiver is rich or poor. The second column shows that receivers punish

Condition	Give to poor	Give to rich	Randomize	poor v. rich	poor v. rand	rich v. rand
All	0.47	0.07	0.35	0.000	0.007	0.000
Certain who is rich and poor	0.52	0.07	0.29	0.000	0.000	0.000
Unertain who is rich and poor	0.43	0.06	0.42	0.000	0.881	0.000
Equal wealth, certainly or statistically			0.69			
Equal wealth, certainly			0.64			

Table 8.4.1: Summary statistics for allocator behavior. The table shows the proportion of trials on which allocators gave the CHF 5 prize to the poorer or richer of the two participants, and how frequently they randomized, percentages. The last 3 columns show the p-values of t-tests comparing the various means.

	(1) Allocator gave to poor	(2) Allocator gave to rich	(3) Allocator randomized
Uncertain which receiver is richer	-0.09	-0.01	0.129
	(0.040)**	-0.022	(0.033)***
Observations	420	420	420
Mean of dependent variable	0.474	0.067	0.355

Table 8.4.2: Results for allocator behavior. The table shows the results from a regression of an indicator that the allocator chose to give as specified in the column heading under the indicated wealth conditions of the receivers. Standard errors clustered at the subject level. * Significant at the 10% confidence level, ** Significant at the 5% confidence level, *** Significant at the 1% confidence level.

	(1) Allocator randomized	(2) Allocator randomized	(3) Allocator randomized	(4) Allocator randomized	(5) Allocator randomized	(6) Allocator randomized
Regression on constant if equal wealth	0.686					
	(0.067)***					
Regression on constant if equal wealth, certain		0.643				
		(0.076)***				
Regression on constant if equal wealth, uncertain			0.729			
			(0.069)***			
Regression on constant if unequal wealth				0.189		
				(0.052)***		
Regression on constant if unequal wealth, certain					0.114	
					(0.050)**	
Regression on constant if unequal wealth, uncertain						0.264
						(0.064)***
F-test: cons=0.5	7.58	3.58	10.96	35.91	60.68	13.58
P-value	0.009	0.067	0.002	0	0	0.001
Observations	140	70	70	280	140	140
Mean of dependent variable	0.69	0.64	0.73	0.19	0.11	0.26

Table 8.4.3: Determinants of randomization by allocators. The table shows the results from a regression of an indicator that the allocator chose to randomize in each of the various situations where there was, or was not, a clearly poorer receiver. An F-test that randomization was the more/less common choice is presented. Standard errors clustered at the subject level. * Significant at the 10% confidence level, ** Significant at the 5% confidence level, *** Significant at the 1% confidence level.

Condition	Give to me	Give to other	Random
All	0.96	-0.65	0.01
Receiver was poor	0.98	-0.97	-0.22
Receiver was poor, certain	0.98	-0.92	-0.19
Receiver was poor, uncertain	0.98	-1.01	-0.25
Equal wealth	0.9	-0.68	0.26
Receiver was rich	1.01	-0.29	0
Receiver was rich, certain	0.96	-0.25	0.06
Receiver was rich, uncertain	1.06	-0.34	-0.05

Table 8.4.4: Summary statistics for receiver behavior. The table shows the average reward (>0) or punishment (<0) that receivers dealt to allocators, contingent on their own initial endowment and the allocator's decision about whom to give the CHF 5 prize or whether to randomize.

the allocator when she chooses to give to the other receiver. Both poor and rich receivers punish the allocator for giving to the other, but poor receivers punish around 3 times as much. Receivers also tend to punish the allocator at relatively high levels when the wealth distribution is equal but the Allocator chooses to give to the other receiver. The final column shows that, on average, poor receivers punish the allocator for choosing to allocate the payment randomly while rich receivers neither punish nor reward the allocator for choosing randomly. Receivers do seem to favor randomization, as reected by a positive reward, when the wealth distribution is equal.

Table 8.4.5 assesses whether these observed patterns and differences are statistically signicant. We estimate the following equation:

$$a_i = \beta_1 R_i + \beta_2 P_i + \varepsilon_i,$$

where a_i is the reward or punishment awarded by receiver i to the allocator, R_i is an indicator that the receiver is rich, and P_i is an indicator that the receiver is poor. The inclusion of indicator variables for being both the rich and poor receiver lends these coe¢ cients the interpretation as the di¤erence in reward and punishment for the rich and poor receivers relative the average reward or punishment in the situation where receivers have equal wealth. We estimate this regression separately for each possible action of the allocator: when the payment is awarded to receiver i, when the payment is awarded to the other receiver and when the allocation is randomly chose.

The first panel, derived from rewards and punishments from receivers who received the payment, confirms that there is no statistical di¤erence in the reward to the allocator between rich and poor receivers when they receive the

	Allocator gives to me		Allocator gives to other		Allocator randomzies	
	(1)	(2)	(3)	(4)	(5)	(6)
	Reward/punishment to allocator	Reward/punishment to allocator	Reward/punishment to allocator	Reward/punishment to allocator	Reward/punishment to allocator	Reward/punishment to allocator
Receiver was poor(=1)	0.079	0.1	-0.286	-0.236	-0.475	-0.421
	(0.085)	(0.104)	(0.113)**	(0.132)*	(0.117)***	(0.133)***
Recevier was rich(=1)	0.107	0.079	0.389	0.436	-0.254	-0.179
	(0.105)	(0.162)	(0.153)**	(0.204)**	(0.109)**	(0.101)*
Uncertain		0.043		0.007		0.043
		(0.08)		(0.082)		-0.121
Receiver poor x uncertain		-0.043		-0.1		-0.107
		(0.126)		(0.121)		-0.204
Receiver rich x uncertain		0.057		-0.093		-0.15
		(0.174)		(0.139)		-0.149
F-test: rich=-poor	1.47		0.39		9.87	
P-value	0.229		0.533		0.002	
Observations	840	840	840	840	840	840
Mean of dep. var.	0.96	0.96	-0.65	-0.65	0.01	0.01

Table 8.4.5: Determinants of reward/punishment by receivers. The table shows the results from a regression of the reward/punishment that the receiver assigns to the allocator for the action indicated in the panel heading, on indicators of the particular conditions under which the allocation was made. Standard errors clustered at the subject level. * Significant at the 10% confidence level, ** Significant at the 5% confidence level, *** Significant at the 1% confidence level..

payment. The second panel conrms that poor receivers do punish allocators that give to the other receiver more than in the equal wealth condition (by 0.28 francs, signicant at the 5% confidence level). Rich receivers punish the allocator less than in the equal wealth condition when the allocator gives the payment to the other, poorer, receiver - by 0.39 francs (statistically different than zero at the 5% confidence level). We fail to reject that the additional punishment by poor receivers when the reward goes to the rich receiver is more or less than the reduced punishment given by richer receivers when the payment goes to the poorer receivers. In the final panel, we show that when wealth is unequal, both poor and rich receivers reward the allocator less for choosing to allocate randomly than in the equal wealth condition: poor receivers reward 0.48 francs less (resulting in an actual punishment relative to the average reward of 0.26 francs in the equal wealth condition) while rich receivers reward 0.25 francs less, the difference between poor and rich receivers is statistically signicant at less than the 1% confidence level.

In the second column we introduce interaction effects, allowing punishments to depend on whether it is certain or uncertain which receiver is richer. We do not find that this matters much in determining the level of reward or punishment.

8.4.3 Estimates of Others' Preferences

In addition to asking receivers how they would reward or punish allocators for various allocation decisions, we asked participants how much they thought other receivers would reward or punish allocators for their choices. Tables 8.4.6 and 8.4.7 show parallel results as those discussed pertaining to receivers actual actions. While the magnitude of the estimated rewards and punishments for other receivers are larger than the actual rewards and punishments inicted by receivers, the relative magnitudes are quite similar to those described pertaining to actual rewards and punishments. One exception is that participants expected rich receivers to reward allocators for giving to them much more than they actually did. But participants correctly guessed that poor receivers would punish more when they did not receive the payment, and rich receivers would punish less when they did not receive the payment. They further correctly assumed that poor receivers would punish more for random allocations than rich receivers. Contrary to the actual behavior of receivers, however, participants thought that uncertainty about which receiver was rich would lessen the punishment inflicted on allocators when they chose to allocate randomly.

Condition	Est: Give to me	Est: Give to other	Est: Random
All	1.6	-0.91	0.16
Receiver was poor	1.59	-1.2	-0.14
Receiver was poor, certain	1.57	-1.24	-0.15
Receiver was poor, uncertain	1.61	-1.16	-0.13
Equal wealth	1.45	-0.93	0.45
Receiver was rich	1.75	-0.6	0.18
Receiver was rich, certain	1.84	-0.65	0.17
Receiver was rich, uncertain	1.67	-0.55	0.18

Table 8.4.6: Summary statistics for estimates of others' behavior. The table shows participants' incentivized estimates of the average reward (>0) or punishment (<0) that receivers dealt to allocators, contingent on their own initial endowment and the allocator's decision about whom to give the CHF 5 prize or whether to randomize.

	Allocator gives to me		Allocator gives to other		Allocator randomzies	
	(1)	(2)	(3)	(4)	(5)	(6)
	Est: Reward/punishment to allocator	Est: Reward/punishment to allocator	Est: Reward/punishment to allocator	Est: Reward/punishment to allocator	Est: Reward/punishment to allocator	Est: Reward/punishment to allocator
Receiver was poor(=1)	0.136	0.086	-0.264	-0.319	-0.586	-0.676
	-0.102	-0.107	(0.079)***	(0.111)***	(0.096)***	(0.113)***
Recevier was rich(=1)	0.3	0.357	0.336	0.271	-0.271	-0.357
	(0.102)***	(0.092)***	(0.135)**	(0.148)*	(0.098)***	(0.111)***
Uncertain		-0.052		-0.029		-0.162
		-0.057		-0.078		(0.065)**
Receiver poor x uncertain		0.1		0.11		0.181
		-0.109		-0.118		(0.100)*
Receiver rich x uncertain		-0.114		0.129		0.171
		-0.093		-0.121		(0.090)*
F-test: rich=poor	6.13		0.23		14.7	
P-value	0.013		0.632		0	
Observations	1260	1260	1260	1260	1260	1260
Mean of dep. var.	1.6	1.6	-0.91	-0.91	0.16	0.16

Table 8.4.7: Estimates of determinants of reward/punishment by receivers. The table shows the results from a regression of the estimated reward/punishment that subjects guess receivers would assign to the allocator for the action indicated in the panel heading on indicators of the particular conditions under which the acllocation was made. Standard errors clustered at the subject level. * Significant at the 10% confidence level, ** Significant at the 5% confidence level, *** Significant at the 1% confidence level..

8.5 Discussion

The use of randomization in medical trials, and more recently in social science research, is scientifically necessary - but may carry ethical costs. Similarly, the use of randomization to allocate scarce goods, spots in a prestigious public school for example, may be cheap and expedient, but is not necessarily fair. While it is difficult to make general statements about whether the social benets of randomization (e.g. the development of useful scientic knowledge) outweigh the costs of perceived unfairness or other ethical costs, the facets of preferences illuminated in this experiment provide some guidance about how to limit the ethical downside when choosing an allocation mechanism. Firstly, we note that those responsible for the allocation of indivisible goods express different preferences over the proper allocation mechanism than, demographically similar, receivers of these goods. Therefore minimizing the "ethical cost" of a particular allocation mechanism requires recognizing that the ethical judgements of receivers are not identical to those making the allocation decision and that the preferences of each group must be taken into account. Secondly, there is a general perception that goods should be allocated to those individuals who most "need" them, suggesting that proper targeting is key to limiting the ethical downside of allocation decisions. Targeting, however, is often expensive (it may be costly, for example locate individuals with the most severe cases of a disease to participate in a medial trial, or to find the poorest households to participate in a school lottery or in a welfare program). We propose a novel approach for measuring the social value of targeting which, although the results are specic to this contest, may be generalized to other settings.

Given that a desire for equality is a well-documented phenomenon (Dawes et al., 2007; Cruces et al., 2011; Ruström & Williams, 2000), it is perhaps not surprising that those in charge of allocating a good have a preference to direct that good to those that most "need" it. In the particular context of this laboratory experiment, we observe that allocators favor awarding the payment to the poorer receiver. This preference for equality is reected to some extent by receivers: wealthy receivers punish allocators who gave the payment to the other receiver less than poor receivers. What is more surprising is that receivers and allocators differ in their perception of appropriate allocation mechanism in more convoluted, and realistic, situations. When there is some uncertainty about which receiver is in fact more "needy" (even if one is clearly more so in expectation) allocators nd random allocation to be a less objectionable form of allocation, whereas receivers preferences over allocations (expressed by the extent to which they reward or punish the allocator) are una¤ected by the introduction of this uncertainty. Thus we note that

the perceived goodness of certain allocation mechanisms can differ between those allocating and those receiving a scarce good. We further note that this is not an "intrinsic" difference, but purely situational as both allocators and receivers were drawn from the same demographic distribution in this experiment. Therefore, in assessing whether any particular allocation mechanism is likely to carry significant ethical costs (and evaluating whether these costs are less than anticipated benets), it is important to understand the preferences and perceptions of all interested parties, recognizing that preferences and perceived costs are shaped by ones position in the transaction.

As noted above, randomization is often a necessary and useful tool for policy makers and researchers, in allocating scarce goods and generating robust scientic knowledge. Randomization, however, is subject to certain ethical concerns as it creates a situation where "some people get it and some people dont." A common response to this objection is that nearly every situation involves scarcity, thus randomization is a fair, as well as efficient or scientically useful, manner of distributing goods. Our experiment suggests that this is true, but only under certain conditions. When there is an equal distribution of wealth between receivers, randomization is generally the preferred allocation mechanism: it is chosen mechanism on the part of allocators and the unique situation where receivers reward allocators for choosing to distribute the payment to a randomly selected receiver. This premise does not appear true, however, when there is disparity in wealth among the receivers - thus properly targeting individuals with equivalent needs appears essential in limiting the ethical costs of random allocation. As noted, however, proper targeting can be expensive. We propose estimating the social value of additional targeting, which can be compared to the costs of targeting, by determining the willingness to pay for additional targeting.

As an example, we note that, in the context of this experiment, punishments for the allocator are socially inefficient since both parties pay for them; thus the average cost to receivers for punishing the allocator is a measure of the social cost of that action. We find that the average cost to receivers (the sum of the absolute values of rewards and punishments) is 1.30 when the wealth distribution is unequal (this figure is the average cost weighted by the frequency with which the allocator chose each allocation option), whereas this cost is only 0.85, or 35% less, when the wealth distribution is equal. This figure suggests that receivers would give up 35% of the average cost in the unequal condition, or 6% of their endowment, to change their situation to one in which the wealth of the potential receivers was equalized - perhaps through better targeting. Such an exercise could be used to determine whether the additional cost of targeting a pool of equally deserving potential recipients of a good outweighs the cost of identifying that group.

Even when randomization is not a desired allocation mechanism, and the goal is simply to provide goods to the most needy population, identifying that population can be difficult and costly. This begs the question of how much should be dedicated to targeting (which often takes away from funds that could be provided as goods to intended recipients). For example, when distributing subsidized food in an impoverished area, how much of the program budget should be spent on identifying the poorest households? Taking into account that less targeting and greater uncertainty about which recipients are poorer means more food distributed in total. To answer such questions, we propose measuring the willingness to pay for better targeting. In the context of our experiment, we note that allocators do not strictly maximize their individual payoff in this experiment: doing so would have implied giving the payment to the poor receiver in all cases, whether there was certainty about their relative wealth or not. Allocators do give the payment to the poorer receiver on average, but they are more likely to randomize when there is uncertainty about which receiver is richer (we also note that participants were fairly good at guessing the average reward or punishment, suggesting allocators would have had a sense of their likely payoff). The increased use of randomization in the presence of uncertainty reduced allocators average payoff from 0.34 to 0.16, or by 44%. While the magnitude is of course specific to this particular context, we note it is relatively large on a percentage basis, and to the allocators endowment of 8 CHF. This suggests a relatively high willingness to pay for additional information about potential recipients, allowing allocators to direct resources to those whom they prefer to reach.

In summary, we find that both allocators and receivers view randomized allocation of resources unfavorably. Most notably, receivers are willing to give up resources to inflict costly punishment on allocators who choose randomization; this behavior is a lower bound on the social costs of random allocation. In addition, we find that allocators and receivers differ in their assessment of the conditions under which random allocation is fair: allocators shift towards random allocation when the incomes of receivers are similar, compared to the situation where incomes are different. receivers, in contrast, do not make this distinction. Thus, avoiding the social costs of random allocation require taking into account the preferences of receivers, which may not be reflected in those of policy makers.

Bibliography

[1] Arrow, K. (1963). Social Choice and Individual Values (2nd ed.). New Haven: Yale University Press. (Original work published 1951).

[2] Bentham, J. (1907). An Introduction to the Principles of Morals and Legislation. Oxford: Clarendon Press. (Original work published 1789).

[3] Bhargava, A. (2008). Randomized controlled experiments in health and social sciences: some conceptual issues. Economics and Human Biology, 6(2), 293-298. doi:10.1016/j.ehb.2008.01.001

[4] Burtless, G. (1995). The Case for Randomized Field Trials in Economic and Policy Research, 9(2), 63-84.

[5] Cruces, G., Truglia, R. P., & Tetaz, M. (2011). Biased Perceptions of Income Distribution and Preferences for Redistribution: Evidence from a Survey Experiment. IZA Discussion Paper No. 5699.

[6] Dawes, C.T., Fowler, J.H., Johnson, T., McElreath, R., & Smirnov, O. (2007). Egalitarian motives in humans. Nature, 446, 794-796.

[7] Eckhoff, T. (1989). Lotteries in allocative situations. Social Science Information/sur les sciences sociales;Social Science Information/sur les sciences sociales, 28(1), 5-22. doi:10.1177/053901889028001001

[8] Erez, E. (1985). Random Assignment, the Least Fair of Them All: Prisoners'attitudes Toward Various Criteria of Selection. Criminology, 23(2), 365-379. doi:10.1111/j.1745-9125.1985.tb00341.x

[9] Fehr, E., Kirchsteiger, G., & Riedl, A. (1998). Gift exchange and reciprocity in competitive experimental markets. European Economic Review, 42(1), 1-34. doi:10.1016/S0014-2921(96)00051-7

[10] Fienberg, S. E. (1971). Randomization and social affairs: the 1970 draft lottery. Science (New York, N.Y.), 171(3968), 255-261. doi:10.1126/science.171.3968.255

[11] Fisher, A. C. (1969). The Cost of the Draft and the Cost of Ending the Draft. The American Economic Review, 59(3), 239-254.

[12] Fisher, R. A. (1928). Statistical Methods for Research Workers (2nd ed.). London: Oliver and Boyd.

[13] Freedman, B. (1987). Equipoise and the ethics of clinical research. The New England Journal of Medicine, 317(3), 141-145. doi:10.1056/NEJM198707163170304

[14] Harrison, G. W., & List, J. A. (2004). Field Experiments. Journal of Economic Literature, 42(4), 1009-1055. doi:10.1257/0022051043004577

[15] Harsanyi, J. C. (1955). Cardinal Welfare, Individualistic Ethics, and Interpersonal Comparisons of Utility. Journal of Political Economy, 63(4), 309-321.

[16] Hillis, J. W., & Wortman, C. B. (1976). Some determinants of public acceptance of randomized control group experimental designs. Sociometry, 39(2), 91-96.

[17] Innes, J. M. (1979). Attitudes towards randomized control group experimental designs in the field of community welfare. Psychological Reports, 44(3 Pt 2), 1207-1213.

[18] Johnson, N., Lilford, R. J., & Brazier, W. (1991). At what level of collective equipoise does a clinical trial become ethical? Journal of Medical Ethics, 17(1), 30-34.

[19] Lilford, R. J., & Jackson, J. (1995). Equipoise and the ethics of randomization. Journal of the Royal Society of Medicine, 88(10), 552-559.

[20] Lockwood, M., & Anscombe, G. E. M. (1983). Sins of Omission? The Non-Treatment of Controls in Clinical Trials. Proceedings of the Aristotelian Society, Supplementary Volumes, 57, 207-227.

[21] MacAtasney, G. (1998). Leitrim and the croppies, 1776-1804 (p. 19). Carrick-on-Shannon: Carrick-on-Shannon and District Historical Society.

[22] Newman, J., Rawlings, L., & Gertler, P. (1994). Using Randomized Control Designs in Evaluating Social Sector Programs in Developing Countries. The World Bank Research Observer, 9(2), 181 -201. doi:10.1093/wbro/9.2.181

[23] Orne, M. T. (2002). On the social psychology of the psychological experiment: With particular reference to demand characteristics and their implications. Prevention & Treatment, 5(1). doi:10.1037/1522-3736.5.1.535a

[24] Rabin, M. (1993). Incorporating Fairness into Game Theory and Economics, 83(5), 1281-1302.

[25] Rawls, J. (1999). A theory of justice. Harvard University Press.

[26] Robbins, L. (1932). An essay on the nature and significance of economic science (p. 15). London: Macmillan.

[27] Rosenblatt, J. R., & Filliben, J. J. (1971). Randomization and the Draft Lottery. Science, 171(3968), 306 -308. doi:10.1126/science.171.3968.306

[28] Ruström, E. E., Williams, M. B. (2000). Entitlements and fairness: an experimental study of distributive preferences. Journal of Economic Behavior & Organization, 43, 75-89.

[29] Sacks, H., Chalmers, T. C. ., & Smith Jr., H. (1982). Randomized versus historical controls for clinical trials. The American Journal of Medicine, 72(2), 233-240. doi:10.1016/0002-9343(82)90815-4

[30] Sen, A. (1970). Collective choice and social welfare. North-Holland Publishing Co. Retrieved from http://www.amazon.com/exec/obidos/redirect?tag=citeulike07-20&path=ASIN/0816277656

[31] Thomas, V. (2010). Evaluation Systems, Ethics, and Development Evaluation. American Journal of Evaluation, 31(4), 540 -548. doi:10.1177/1098214010373648

[32] Toroyan, T., Roberts, I., & Oakley, A. (2000). Randomisation and resource allocation: a missed opportunity for evaluating health care and social interventions. Journal of Medical Ethics, 26(5), 319-322. doi:10.1136/jme.26.5.319

[33] United States v. Holmes, 26 Fed. Cas. 360 (No. 15383) (Cir. Ct. E. Dist. Pa.) (1842).

[34] Zwane, A. P., Zinman, J., Van Dusen, E., Pariente, W., Null, C., Miguel, E., Kremer, M., et al. (2011). Being surveyed can change later behavior and related parameter estimates. Proceedings of the National

Academy of Sciences of the United States of America, 108(5), 1821-1826. doi:10.1073/pnas.1000776108

Chapter 9

Both Sides Retaliate in the Israeli-Palestinian Conflict

9.1 Summary

Ending violent international conflicts requires understanding the causal factors that perpetuate them. In the Israeli-Palestinian conflict, Israelis and Palestinians each tend to see themselves as victims, engaging in violence only in response to attacks initiated by a fundamentally and implacably violent foe bent on their destruction. Econometric techniques allow us to empirically test the degree to which violence on each side occurs in response to aggression by the other side. Prior studies using these methods have argued that Israel reacts strongly to attacks by Palestinians, while Palestinian violence is random (i.e. not predicted by prior Israeli attacks). Here we replicate prior findings that Israeli killings of Palestinians increase after Palestinian killings of Israelis, but crucially show further that when non-lethal forms of violence are considered, and when a larger dataset is used, Palestinian violence also reveals a pattern of retaliation: i) the firing of Palestinian rockets increases sharply after Israelis kill Palestinians, and ii) the probability (although not the number) of killings of Israelis by Palestinians increases after killings of Palestinians by Israel. These findings suggest that Israeli military actions against Palestinians lead to escalation rather than incapacitation. Further, they refute the view that Palestinians are uncontingently violent, showing instead that a significant proportion of Palestinian violence occurs in response to Israeli behavior. Well established cognitive biases may lead participants on each side of the conflict to under-appreciate the degree to which the other side's violence is retaliatory, and hence to systematically underestimate their own role in perpetuating the conflict.

9.2 Introduction

Over half of Israelis and three quarters of Palestinians think the other side seeks to take over their land. When accounting for their own acts of aggression, Israelis often claim to be merely responding to Palestinian violence (1-3), and Palestinians often see themselves as simply reacting to Israeli violence (4-6). Are these views just self-serving rationalizations designed to justify violence committed for other reasons, or are they in part true? That is, do Israeli attacks against Palestinians in fact occur in response to prior violence by Palestinians, and do Palestinian attacks against Israelis occur in response to prior violence by Israelis? The answers to these questions are important because they carry implications about the nature of the actors on each side, and the most effective strategies for reducing the conflict. Fortunately, these questions can be approached empirically by applying quantitative econometric methods (vector autoregression) to well-documented empirical databases of the timeline of violence over the Second Intifada.

Several recent studies have taken this approach (7-10) and argued that Israeli killings of Palestinians fit the pattern of retaliation, increasing immediately after Palestinian killings of Israelis, while Palestinian killings of Israelis do not fit the pattern of retaliation (8, 10). This finding supports the narrative that Israel merely responds to Palestinian violence, whereas Palestinian attacks are not contingent on Israeli behavior, instead reflecting a fundamental and non-negotiable goal of harming Israel. This view in turn bolsters Israeli arguments for military over diplomatic solutions on the grounds that "there is no one to talk to". However, the prior analyses suffer from several limitations, making their conclusions premature.

Most importantly, prior analyses consider only killings, not other forms of violence. Three problems arise as a result. First, multiple nonlethal forms of aggression occur on both sides, such as unsuccessful attacks on Israelis by Palestinians, and house demolitions, imprisonment, blockades, and restrictions of movement by Israel against Palestinians. Any of these nonlethal forms of aggression could either cause or constitute retaliation. Second, vector autoregression asks whether killings of one side follow killings by the other side at a consistent time lag. However, given the lower level of organization and technology of the various armed Palestinian factions, such time-locked responses may be difficult for Palestinians to achieve; Jaeger & Paserman suggest, alternatively, that Palestinians may intentionally randomize the timing of their attacks to preclude preventative measures being taken by Israel, which would render any retaliatory attacks invisible to VAR (see 11-13 for a discussion of whether Palestinian groups act rationally). Third, some crucial factors may be invisible to vector autoregression because they

are not punctate events but chronic conditions: e.g., chronic fear of suicide bombing and/or rocket attacks among Israelis, and the chronic oppression of living under occupation among Palestinians.

These problems illustrate the impossibility of modeling and testing all potential nonlethal causes and forms of retaliation in the Israeli/Palestinian conflict. However, one form of violence avoids all three problems and is therefore amenable to econometric testing: the firing of Qassam rockets from Gaza into Israel. Rocket firings are punctate events that can be precisely timed; they are rarely lethal (between January 2001 and April 2008, the Israeli Defense Forces (IDF) and B'tselem registered 3645 Qassam rocket firings, but only 15 associated fatalities); and precise quantitative data on the numbers of rockets fired every day are available. Together, this means that if rockets are used in a retaliatory fashion this might be evident in a vector autoregression. We therefore conducted analyses of data obtained from the Israeli Defense Forces (IDF) that give the number of daily Qassam rockets fired by Palestinians into Israeli territory, irrespective of whether anyone died as a result. Our analysis covers the period of January 2001 - April 2008, i.e. from the beginning of the Second intifada until the ceasefire that preceded the December 2008 Gaza War.

We find that Palestinian Qassam rocket firings increase sharply on the day following the killing of Palestinians by Israel. In addition, we show that the probability of Palestinian killings of Israelis (although not the number of people killed) increases following the killing of Palestinians by Israel. Thus, it appears that Palestinian violence does contain an element of retaliation, and that Israeli military operations against Palestinians lead to escalation rather than incapacitation. Consistent with previous analyses, we confirm that Israeli aggression, too, contains an element of retaliation, in that Israel is more likely to kill Palestinians on days following killings of Israelis by Palestinians. Together, these results suggest that, contrary to previous analyses that characterized Israeli violence alone as retaliatory, the dynamics of retaliation in the Second Intifada are bidirectional.

9.3 Results

9.3.1 Summary statistics

Figure 9.3.1 shows the timeline of violence between Israelis and Palestinians from January 1, 2001, to April 16, 2008. Each panel is a histogram plotting the daily count of events; panel A shows Palestinian fatalities resulting from Israeli attacks, panel B shows Israeli fatalities resulting from Palestinian

attacks, and panel C shows daily counts of Qassam firings by Palestinians into Israeli territory.

Table 9.3.1 shows summary statistics associated with these variables. Palestinian fatalities, Israeli fatalities, and Qassam firings occurred on 49.75%, 11.85% and 34.66% of all days in the dataset, respectively. There were a total of 4874 Palestinian fatalities, 1062 Israeli fatalities, and 3645 Qassam firings; this corresponds to a daily average of 1.65 ± 3.52 (mean ± standard deviation) Palestinian fatalities, 0.36 ± 1.66 Israeli fatalities, and 1.37 ± 3.75 Qassam firings. Of the Qassam firings, 15 (0.41%) resulted in fatalities, underscoring that Qassam firings are a largely non-lethal form of Palestinian aggression.

In the following we consider two versions of each of the three variables: the number of daily events on one hand, and the daily incidence on the other hand. The former variable is the count of events, i.e. the number of Palestinian and Israeli fatalities and Qassam firings on any given day. The incidence is a dummy variable that takes value one on days when one or more events occurred, and zero otherwise.

Table 9.3.2 shows the pairwise correlation coefficients and their p-values for the three variables, both in terms of number of daily events as well as incidence. It can be seen that Israeli and Palestinian aggression are strongly positively correlated (although Qassam firings and Palestinian fatalities only correlate in levels, not in incidence), while the two forms of Palestinian aggression correlate negatively, potentially reflecting a substitution effect. This cursory first analysis suggests that Israeli and Palestinian violence may be mutually related. However, to test for retaliatory aggression, we must consider relationships across time.

9.3.2 Impulse response functions

To test for retaliatory patterns of violence, in the restricted sense of temporal relatedness proposed by Granger (1969), we initially computed impulse response functions for the three variables. As pointed out by (8), the value of the Israeli response function on day t can be interpreted as the excess number of attacks against Palestinians t days after a Palestinian attack against Israelis, normalized by the number of attacks against Israelis. The Palestinian response function is interpreted analogously.

Figures 9.3.2 and 9.3.3 show the response functions for levels and incidence, respectively. In each panel, an attack of one party occurs at time 0, and the function plots the excess number (Figure 9.3.2) or probability (Figure 9.3.3) of attacks by the other side occurring on the following days.

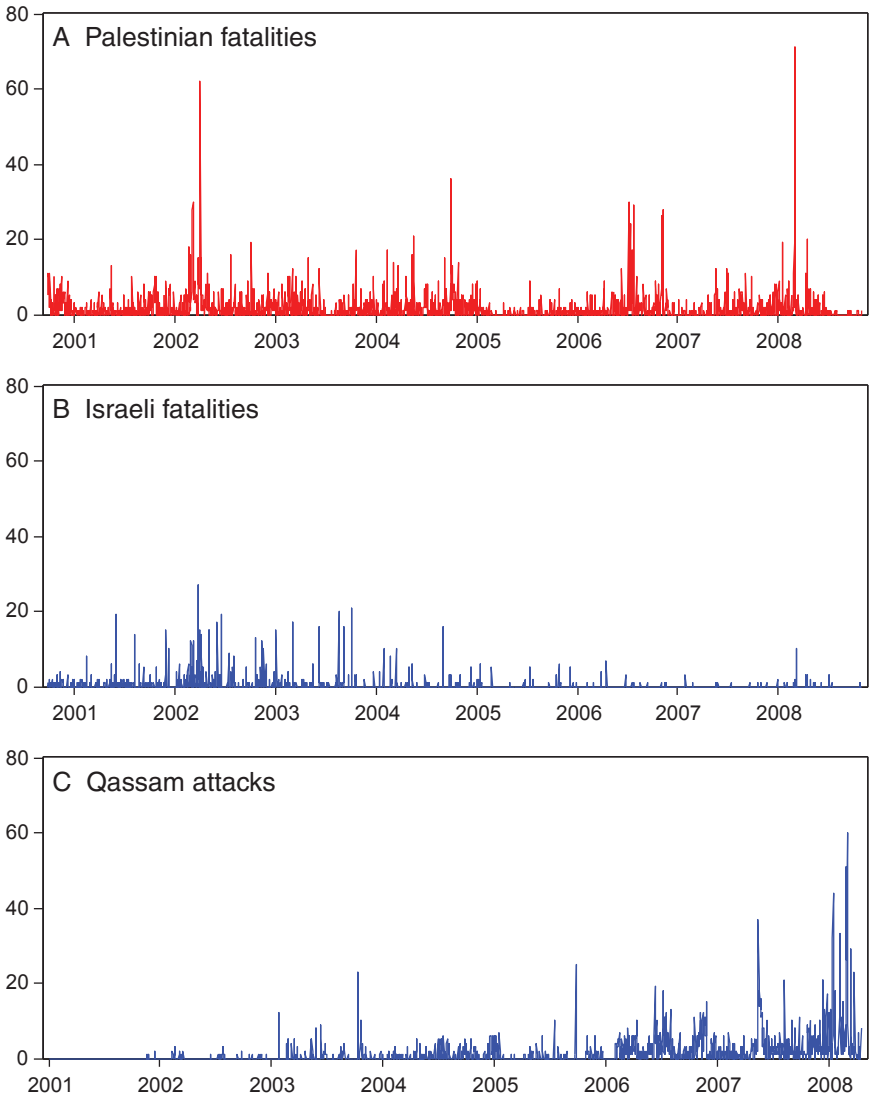

Figure 9.3.1: Timeseries of Palestinian fatalities (A), Israeli fatalities (B), and Qassam attacks by Palestinians on Israel (C). Data are daily event counts between 2000 and 2008 (cf. Table 1 for exact dates), compiled from data from the Israeli human rights organization Bt'selem (A & B) and the Israeli Defense Forces (C).

	(1) Palestinian fatalities	(2) Israeli fatalities	(3) Qassam attacks
Data begins	September 29, 2000	September 29, 2000	January 1, 2001
Data ends	October 29, 2008	October 29, 2008	April 16, 2008
Days in dataset	2953	2953	2663
Number of days with events	1469	350	923
Percentage of days with events	49.75%	11.85%	34.66%
Total number of events	4874	1062	3645
Total number of fatalities	4874	1062	15
Percentage of fatal events	100%	100%	0.41%
Minimum events per day	0	0	0
Maximum events per day	71 (March 1, 2008)	27 (March 27, 2002)	60 (March 1, 2008)
Mean events per day	1.65	0.36	1.37
Standard deviation	3.52	1.66	3.75

Table 9.3.1: Summary Statistics. Table reports summary statistics for Palestinian fatalities (column 1), Israeli fatalities (column 2), and Qassam attacks by Palestnians on Israel (column 3). The data were obtained from the Israeli Human Rights Bt'selem (columns 1-2) and the Israeli Defense Forces (column 3).

Variable pair		Levels	Incidence
Palestinian x Israeli fatalities	Coefficient	0.1092	0.1449
	p-value	0.0000	0.0000
Palestinian fatalities x Qassam attacks	Coefficient	0.2708	0.0286
	p-value	0.0000	0.1399
Qassam attacks x Israeli fatalities	Coefficient	-0.0405	-0.0913
	p-value	0.0367	0.0000

Table 9.3.2: Pairwise correlations. Shown are correlation coefficients for Palestinian and Israeli fatalities and Qassam firings, and their associated p-values. Significant correlations are set in bold.

Our main question was whether Israeli and Palestinian violence show retaliatory patterns. Specifically, we hypothesized that Qassam rocket firings might to some extent be a consequence of previous Israeli aggression against Palestinians. Figure 9.3.2D suggests that this may be the case: the number of Qassam rocket firings by Palestinians increases compared to baseline on the days following Israeli killings of Palestinians. Interestingly, Israel does not appear to respond to firings of Qassam rockets (Figures 9.3.2C, 9.3.3C). Conversely, Figures 9.3.2A and 9.3.3A suggest that, as reported previously (8), Israeli killings of Palestinians increase on the days following killings of Israelis by Palestinians. In contrast to previous findings, however, Palestinians appear to not only respond to Israeli attacks with Qassam rockets, but also by increasing the incidence of killings of Israelis.

9.3.3 Statistical model

To quantify these results, we employed a standard vector autoregression model (VAR), in which current Israeli and Palestinian fatalities are regressed on lagged values of both variables. We perform these regressions twice for each pair of variables: once for levels, and once for the dummy incidence variables. The results of these regressions for the three variables under consideration are shown in Supp. Tables 4-7.

To test based on these regressions whether Israeli attacks predict Palestinian attacks and vice-versa, we computed the F-statistics for the joint significance of the lagged coefficients of the respective other variable. The results are shown in Table 9.3.3. They confirm the patterns observed in Figures 9.3.2 and 9.3.3: past Palestinian fatalities significantly predict an increase in firings of Qassam rockets, both in terms of the number of rockets fired, as well as in terms of the number of days on which firings occur. Thus, the firing of Qassam rockets appears to occur at least partly in response to previous

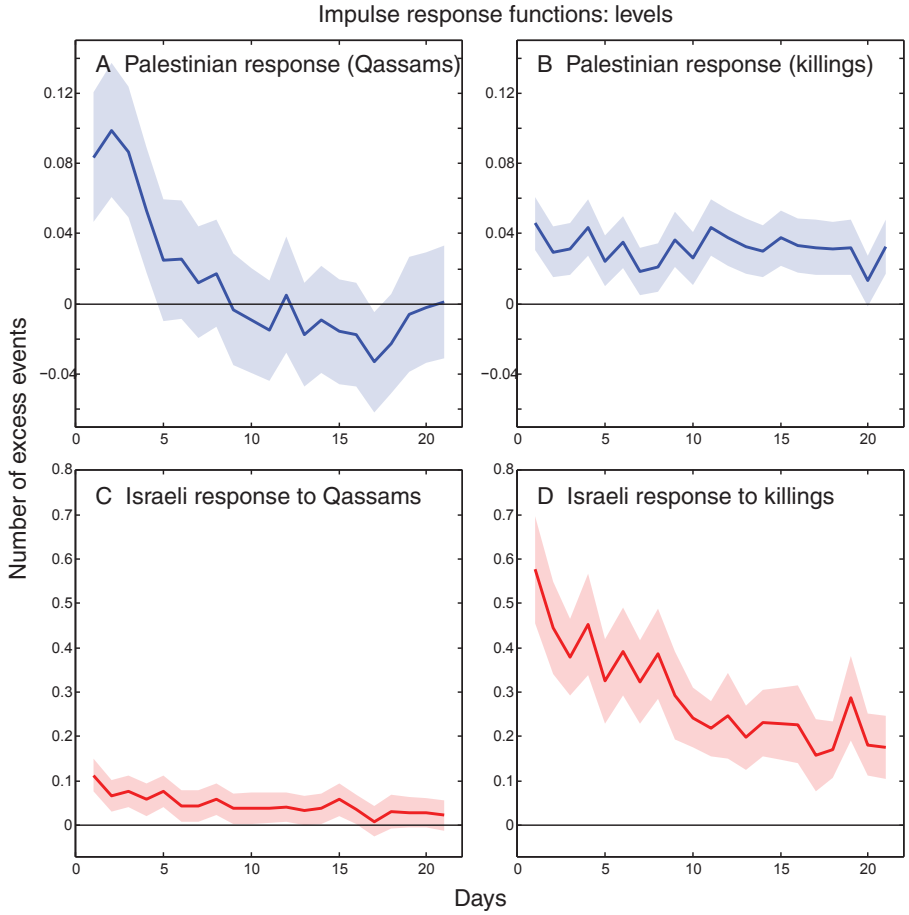

Figure 9.3.2: Impulse response functions for number of events (levels). Each graph shows the excess number of attacks by one side following an attack from the respective other side. (A) shows the Palestinian response in terms of Qassam rocket attacks to killings of Palestinians by Israel. (B) shows the Palestinian response in terms of killings of Israelis to killings of Palestinians by Israel. (C) shows the Israeli response in terms of killings of Palestinians to Qassam attacks by Palestinians. Finally, (D) shows the Israeli response in terms of killings of Palestinians to killings of Israelis by Palsetinians.

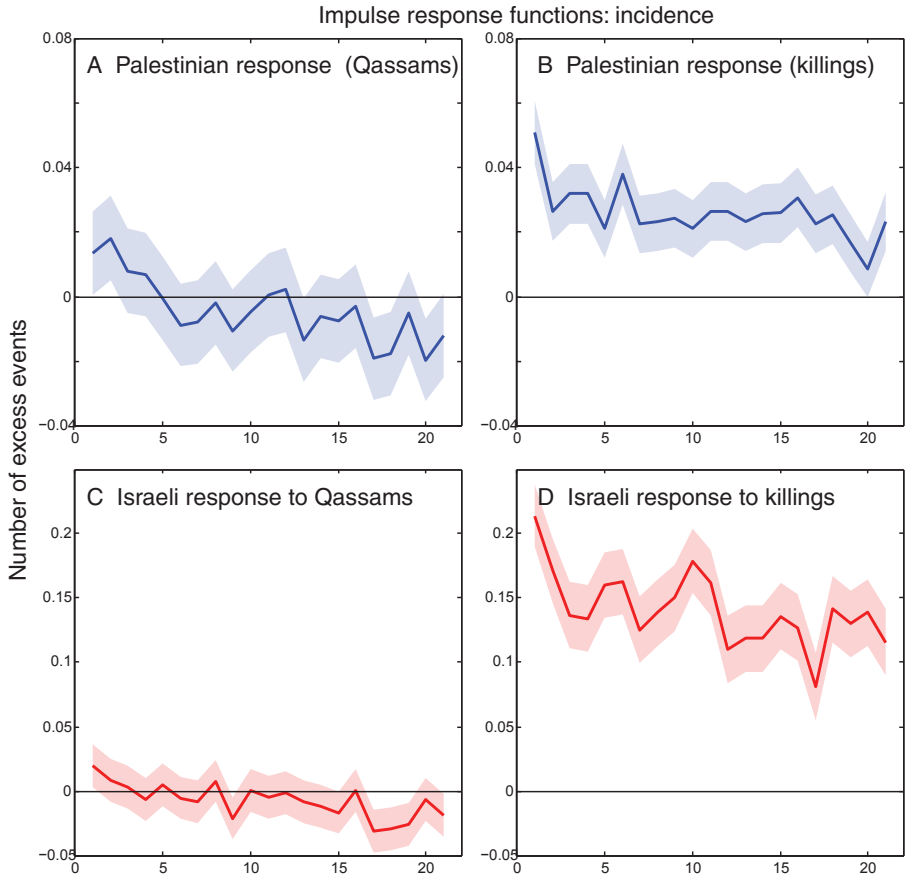

Figure 9.3.3: Impulse response functions for probability of events (incidence). Each graph shows the excess probability of attacks by one side following an attack from the respective other side. (A) shows the Palestinian response in terms of Qassam rocket attacks to killings of Palestinians by Israel. (B) shows the Palestinian response in terms of killings of Israelis to killings of Palestinians by Israel. (C) shows the Israeli response in terms of killings of Palestinians to Qassam attacks by Palestinians. Finally, (D) shows the Israeli response in terms of killings of Palestinians to killings of Israelis by Palsetinians.

Israeli attacks on Palestinians. (Note that this analysis also shows that the small increase in the incidence of Qassam firings following killings of Palestinians shown in Figure 9.3.3D is statistically significant when controlling for the history of attacks.)

In addition, in contrast to the results of (8), we find that past Palestinian fatalities also predict an increase in the probability that Palestinians will kill any Israelis in the following days (incidence).

Conversely, we find that killings of Palestinians by Israelis also contain a retaliatory element, both in terms of levels and incidence; however, this retaliation occurs only after killings of Israelis, and not after (mostly non-lethal) Qassam firings.

To understand the magnitude of these effects, we estimated the percentage of attacks that can be ascribed to retaliation. Our results suggest that the number of Qassams fired increases by 6% on the first day after a single killing of a Palestinian by Israel, the probability of any Qassams being fired increases by 11%, and the probability of any Israelis being killed by Palestinians increases by 10% (for details see Materials & Methods). Conversely, one day after the killing of a single Israeli by Palestinians, the number of Palestinians killed by Israel increases by 9%, and the probability of any Palestinians being killed by 20%.

We can then use these values to estimate what proportion of aggression on either side can be attributed to prior attacks from the other side throughout the period under consideration. This calculation (see Materials & Methods) showed that retaliation accounts for a larger fraction of Palestinian compared to Israeli aggression: in the levels specification, 10% of all Qassam rockets (358 in number) can be attributed to prior Israeli attacks on Palestinians, but only 4% of killings of Palestinians by Israel (158 in number) can be attributed to prior Palestinian attacks on Isarel. In the incidence specification, 6% of all days on which Palestinians attack Israel with rockets, and 5% of all days on which they attack by killing Israelis, can be attributed to retaliation; in contrast, this is true for only 2% of all days on which Israel kills Palestinians.

To solidify these results, we conducted several controls. First, to ascertain that the increase of Qassam attacks following Palestinian fatalities is not due to Palestinian fatalities on the same day (which might have occurred after the Qassam attacks on that day), we added a control variable for same-day Palestinian fatalities (Table 9.3.3); this does not alter the results. Second, we wished to control for the elevated level of Qassam attacks on day 0, i.e., concurrent with Palestinian fatalities: Qassam attacks on the following day(s) might be the result of previous Israeli aggression, which, however, might in turn be triggered by these Qassam attacks on day 0. We address this potential confound in two ways. First, the VAR analysis controls for previous own

		(1) Palestinian retaliation using Qassams		(2) Palestinian retaliation using killings		(3) Israeli retaliation for Qassams		(4) Israeli retaliation for killings	
Specification	Test statistic	Levels	Incidence	Levels	Incidence	Levels	Incidence	Levels	Incidence
Basic	F	**3.0733**	**2.3693**	1.7486	**8.8439**	1.0363	1.1364	**2.9971**	**10.3071**
	p	**0.0090**	**0.0372**	0.1201	**0.0000**	0.4139	0.2982	**0.0176**	**0.0000**
Control same-day events	F	**3.5771**	**2.2527**	0.8238	**5.4941**	**1.6899**	1.2655	**2.7133**	**7.5588**
	p	**0.0032**	**0.0467**	0.5325	**0.0000**	**0.0233**	0.1824	**0.0285**	**0.0000**
Omit mutual events at t-1	F	1.4334	**2.4531**	0.5007	**7.6371**	1.2465	1.2170	**2.1231**	**5.5271**
	p	0.2090	**0.0317**	0.7759	**0.0000**	0.1973	0.2216	**0.0754**	**0.0002**
Control variable for years	F	**3.125**	**3.824**	1.1177	**3.4432**	0.9271	**2.0559**	**2.8322**	**4.5972**
	p	**0.0081**	**0.0019**	0.3486	**0.0042**	0.5584	**0.0026**	**0.0233**	**0.0011**

Table 9.3.3: Israeli and Palestinian relaliation for killings and Qassam attacks. Table reports the test statistics for the test of the null hypothesis that the lagged coefficients on the respective other variable are jointly equal to zero. Significant statistics are set in bold and can be interpreted as retaliation by one party for previous violence from the other side. Columns 1-2 report Palestinian retaliation after killings of Palestinians by Israel, i.e. killings of Israelis by Palestinians (column 1), or Quassam attacks by Palestinians on Israel. Columns 3-4 report Israeli retaliation, i.e. killings of Palestinians by Israel, following either Qassam attacks by Palestinians on Israel (column 3), or killings of Israelis by Palestinians (column 4).

aggression; thus, Qassam attacks are higher following Palestinian fatalities, holding constant previous Qassam attacks. Second, as can be seen from the regression tables (Suppl. Tables 4-7), the increase of Qassam rockets after Palestinian fatalities was most significant on the first day after such fatalities. We therefore repeated the analysis without taking into consideration attacks which occurred on days after mutual Israeli and Palestinian attacks. The results are shown in Table 3; again it can be seen that Palestinian fatalities predict an increase in Qassam attacks, but not vice-versa, although note that the effect reaches significance only in the incidence specification.

9.4 Discussion

In this study we replicate prior findings that Israeli killings of Palestinians fit the pattern of retaliation, that is, they increase after Palestinian killings of Israelis (8, 10; see also 14-18). However, unlike prior studies, we show that Palestinian violence also shows a retaliatory pattern: i) the firing of Qassam rockets increases sharply after Israelis kill Palestinians, and ii) the probability (although not the number) of killings of Israelis by Palestinians increases after killings of Palestinians by Israel. This result argues against the narrative that sees Palestinians as inherently and unconditionally violent. Instead, our analysis shows that Palestinian violence is in part contingent on Israeli violence: Palestinians, like Israelis, are more likely to attack after they themselves have been attacked. In addition, it shows that Israeli military actions against Palestinians may lead to escalation of violence rather than incapacitation of Palestinian military operations against Israel.

Is the Israeli/Palestinian conflict a "cycle of violence", in which each attack is followed by a counterattack in a tit-for-tat fashion? Jaeger & Paserman (8) argue that it is not, because their analyses found that Israeli violence fit a pattern of retaliation whereas Palestinian violence did not (see also 19-21, 9). In contrast, our data show that both sides retaliate, consistent with "tit for tat" dynamics. One might argue that the firing of rockets does not constitute the continuation of a cycle, since rocket attacks rarely lead to Israeli fatalities. Indeed, our data show that rocket attacks are usually not followed by retaliation by Israel. Nonetheless, rocket attacks cause widespread public anger in Israel and attract broad media coverage. Thus, even though rocket attacks are usually not met with an immediate, time-locked increase in killings of Palestinians, they may nonetheless lead to an overall (not time-locked) increase in Israeli violence against Palestinians, thus continuing the cycle in the longer run. In addition, we find that Palestinian retaliation also occurs through an increased probability of killings of Israelis;

this Palestinian response is unambiguously a continuation of the cycle of violence, both in terms of public attention and Israeli military reaction. Thus our data show that, in contrast to prior reports, some aspects of the Israeli Palestinian conflict fit the tit for tat "cycle of violence" pattern (see also 15, 22-24).

Our results may be most newsworthy to the participants in this conflict themselves. Palestinians and Israelis each tend to describe themselves as victims in the conflict, each side describes their own violent attacks as retaliatory, and each describes the attacks of the other side as caused by aggressive intent rather than as responses to external attack. Of course, public statements from each side are bound to reflect self-serving efforts to gain the moral high ground in the battle of public opinion. However, deeper cognitive forces may also be at play here (25): It is well documented in the psychology literature that people tend to see their own behavior as driven more by the situation they are in (e.g., being under attack by the other), but to see the behavior of others as driven more by their disposition (e.g., being inherently hostile or violent; 26). These fundamental cognitive biases may lead each side to underappreciate the degree to which the other side's violence is retaliatory, and therefore to systematically underestimate their own role in perpetuating the conflict (see also 27).

Despite the symmetrical pattern of violence we report here, in which both sides retaliate for attacks from the other side, other aspects of the Israeli-Palestinian conflict are characterized by deep asymmetries. Most obviously, over four times as many Palestinians as Israelis were killed in the period investigated here; during the subsequent 2008-2009 Gaza war (after the period studied here) this asymmetry reached about 100 Palestinian deaths for each Israeli death. Second, Israel controls and often severely restricts many facets of Palestinian life, including access to food and medicine and freedom to move within and outside their territory, whereas Palestinians exert no such control over Israelis. Third, in an asymmetry particularly importantly for the present study, the Israeli Defense Forces is one of the most technologically sophisticated and well-trained military organizations in the world, whereas the Palestinians have no regular military at all, only an array of armed factions not under direct central governmental control. Indeed, it is precisely this lack of central control and much lower technological sophistication on the Palestinian side that probably explains why prior vector autoregression analyses focusing on only killings did not detect the time-locked pattern of Palestinian retaliation that we found in rocket firings.

Given the fact that Qassam attacks are almost completely ineffective in killing Israelis, one might ask why Palestinians engage in firing these rockets. Several potential answers suggest themselves. One intuitive reason is that

Palestinians use all means available to them to respond to Israeli attacks; the military power of Palestinians is vastly inferior to that of Israel, and Palestinians do not have access to many means other than rockets. (Of course we do not claim that Palestinian retaliation occurs only through rockets or the probability of Israeli fatalities; other forms of Palestinian retaliation may well exist, but data on it may be more difficult to obtain.) In addition, there is some evidence that Israel increases its vigilance (e.g. by closing borders) after it has killed Palestinians (8). This fact might make it even more difficult for Palestinians to retaliate, leading them to resort to rockets as the only viable form of retaliation (28-30). Second, while Qassams are not particularly effective at killing Israelis, they do cause significant psychological distress among Israelis (while not incurring a military response from Israel, discussed below), which is mirrored in the strong political and public response to the rocket attacks in Israel (3, 7). A related possibility is that rocket attacks are used by Palestinian factions (particular extremist ones) to solidify their position and reputation among the population (31-33, 9). Finally, attacks may be used to affect political opinion in Israel (34-36), cause economic damage to Israel (37-38), or more generally to derail the peace process (39-40).

Conversely, another question that emerges from these results is why Israel responds so strongly to killings of Israelis, but does not show a time-locked response in terms of Palestinian fatalities to Qassam rockets over the period under consideration. One possible explanation is that the Israeli Defense Forces recognize that only a relatively small proportion of rocket attacks actually result in fatalities; this fact may reduce the IDF's motives to retaliate against Qassams militarily. Alternatively, one might conjecture that Israel responds to Qassam firings with less-than-lethal violence against Palestinians; we do not have data available that would speak to this question, but future analyses might ask whether Israel responds to Qassam firings by e.g. imposing restrictions on freedom of movement on Palestinians.

An important caveat of the present study is that our analyses cannot test causation directly; rather, they test whether the timeline of events fits the temporal profile expected for retaliation, i.e. whether one event reliably predicts the other event at a later time (41). Nonetheless, the most straightforward interpretation of our results is that it is not only Israelis who retaliate for killings of Israelis by killing Palestinians, but also Palestinians who retaliate for killings of Palestinians by firing rockets and by increasing the probability of killing Israelis on any given day.

A related potential concern regarding our results is reverse causation. Our data could, for instance, reflect killings of Palestinians by Israel in anticipation of rocket attacks, rather than Palestinian retaliation for previous killings

of Palestinians by Israel. Three facts argue against this possibility. First, one would expect that, if Israel kills Palestinians in anticipation of rocket attacks, that these actions would lead to a decrease in rocket attacks following killings of Palestinians by Israel, rather than the increase that is actually observed. Second, if prevention of attacks was the main reason for Israeli attacks, one would expect Israeli killings of Palestinians to occur not only before, but also after rocket attacks; in fact, one might argue that killings of Palestinians by Israel should increase particularly strongly following rocket attacks, reflecting Israeli operations to shut down the cells that were responsible for the attacks. However, we find that killings of Palestinians by Israel actually do not increase significantly following rocket attacks. This finding suggests that the killings of Palestinians by Israel preceding rocket attacks are usually not preventative measures to suppress rocket attacks. Finally, the preceding argument is strengthened by the IDF rules of engagement surrounding Palestinian rocket attacks: according to (3), "[s]oldiers are allowed to fire freely at rocket- and mortar-launching cells immediately before, during or after a launch, and with permission from a senior officer, they can also fire at Palestinians trying to lay bombs within half a kilometer of the border fence. Other than that, however, no offensive operations are permitted." In other words, it is likely that preventative attacks by Israel would be concentrated on the day of the (attempted) rocket attacks, rather than occurring mainly on the day prior. Since the data show that Israeli attacks in fact do occur on the day preceding rocket attacks, we conclude that the rocket attacks are a consequence, rather than a cause, of the Israeli attacks. These arguments hold analogously for the increased probability of killings of Israelis by Palestinians following Israeli attacks on Palestinians. Conversely, the argument against reverse causality in the context of the Israeli response to Palestinian attacks has been made by (8) and (10).

In sum, our analyses of the temporal dynamics of violence in the Second Intifada show evidence both of Israeli retaliation for Palestinian violence, and also Palestinian retaliation for Israeli violence. These findings suggest that the Israeli-Palestinian conflict is characterized by retaliatory dynamics in both directions: both sides respond to killings by the other side in a time-locked fashion; in the case of Israel, this response takes the form of killings of Palestinians, whereas in the case of the Palestinians, it comes in the form of (mostly nonlethal) Qassam attacks against Israel. In addition, Palestinians appear to retaliate by increasing the probability of killing Israelis on any given day. The implication of our results is that both sides are at least to some extent correct when they claim that their aggression occurs in response to previous aggression from the respective other party. To the extent that both sides see themselves in a purely retaliatory role, our data suggest that

in doing so they may under-appreciate the extent to which the violence of the other side is contingent on their own. An increased awareness of this bias may lead both sides to better understand their own role in perpetuating the conflict, and thus contribute to its resolution (27).

9.5 Materials & Methods

Daily counts of Israeli and Palestinian fatalities were obtained from Bt'Selem (www.btselem.org); daily counts of Qassam attacks from the Israeli Defense Forces.

Following (8), we defined the Israeli response function as

$$IsrRF_t = \left(\frac{\sum_{s:I_s>0} I_s}{\sum_{s:I_s>0} 1}\right)^{-1} \left(\frac{\sum_{s:I_{s-t}>0} P_s}{\sum_{s:I_{s-t}>0} 1} - \frac{\sum_s P_s}{T}\right)$$

and the Palestinian reaction function as

$$PalRF_t = \left(\frac{\sum_{s:P_s>0} P_s}{\sum_{s:P_s>0} 1}\right)^{-1} \left(\frac{\sum_{s:I_{s-t}>0} I_s}{\sum_{s:I_{s-t}>0} 1} - \frac{\sum_s I_s}{T}\right).$$

Here, I_s and P_s denote the number of attacks against Israelis and Palestinians on day s, respectively; attacks against Israelis can either be Israeli fatalities or Qassam firings.

To prepare the Vector Autoregression, we first used Schwarz's Bayesian Information Criterion to choose the most appropriate autoregressive order for each of the three variables; the optimal number of lags was 5 for Palestinian fatalities, 4 for Israeli fatalities, and 22 for Qassam rocket firings (see Supp. Information, Supp. Table 1). We then ascertained that the timeseries were stationary using an augmented Dickey-Fuller Test (see Supp. Information, Supp. Table 2). Next we considered two alternative VAR models, namely ordinary least squares (OLS) and negative binomial (NB) models. Leave-one-out crossvalidation showed that OLS made better out-of-sample predictions than NB; we therefore based our analyses on OLS (Supp. Table 3).

Thus, we fit the following system of equations:

$$I_t = \alpha_I + \sum_{s=1}^{L_I} \beta_{I,s} I_{t-s} + \sum_{r=1}^{L_P} \gamma_{I,r} P_{t-r} + \varepsilon_{I,t}$$

$$P_t = \alpha_P + \sum_{r=1}^{L_P} \beta_{P,r} P_{t-r} + \sum_{s=1}^{L_I} \gamma_{I,s} I_{t-s} + \varepsilon_{P,t}.$$

Here, I_t and P_t again denote attacks against Israelis and Palestinians on day t, respectively; L_I and L_P is the optimal number of lags on attacks against Israelis and Palestinians, respectively, determined by information criteria; $\alpha_{I,t}$ and $\alpha_{P,t}$ are the constant terms, and $\varepsilon_{I,t}$ and $\varepsilon_{P,t}$ the error terms.

To test whether past attacks on Palestinians predict current attacks against Israelis, we asked whether the L_I elements of the coefficient vector γ_I are jointly significantly different from zero by computing their F-statistic. Conversely, to test whether past attacks on Israelis predict current attacks against Palestinians, we asked whether the L_P elements of the coefficient vector γ_P are jointly significantly different from zero.

Further details are given in Supporting Information online.

Bibliography

[1] Feldman, N. (2007). Piece Process. New York Times, June 24, 2007. http://www.nytimes.com/2007/06/24/magazine/24wwln-lede-t.html. March 6, 2010.

[2] Fisher, I. (2006). In Gaza, Defiantly Awaiting Israeli Retaliation. New York Times, June 27, 2006. http://www.nytimes.com/2006/06/27/world/middleeast/27gaza.html. March 6, 2010.

[3] Harel, A. (2008). Olmert: We won't just defend against Gaza terror, we'll attack. Ha'aretz, December 9, 2008. http://www.haaretz.com/print-edition/news/olmert-we-won-t-just-defend-against-gaza-terror-we-ll-attack-1.259125. April 20, 2010.

[4] CNN (2008). IDF kills militant as Hamas pounds southern Israel with rockets. CNN, December 24, 2008. http://edition.cnn.com/2008/WORLD/meast/12/24/israel.rockets/index.html. April 20, 2010.

[5] Ezzedeen Al-Qassam Brigades Information Office (2006). 2 Qassam Rockets Fired at Kissofim Checkpoint. Ezzedeen Al-Qassam Brigades Information Office, June 14, 2006. http://www.qassam.ps/statement-2-2_Qassam_Rockets_Fired_at_Kissofim_Checkpoint.html. April 20, 2010.

[6] Ezzedeen Al-Qassam Brigades Information Office (2008). E.Q.B. fired (3) Qassam rockets at the Zionist military bases as a response to the Zionist massacre in Gaza. Ezzedeen Al-Qassam Brigades Information Office, December 27, 2008. http://www.qassam.ps/statement-1241.html. April 20, 2010.

[7] Jaeger, D.A., & Paserman, M.D. (2006). Israel, the Palestinian Factions, and the Cycle of Violence. American Economic Review, 96, 45–49.

[8] Jaeger, D.A., & Paserman, M.D. (2008). The Cycle of Violence? An Empirical Analysis of Fatalities in the Palestinian-Israeli Conflict. American Economic Review, 98, 1591–1604.

[9] Jaeger, D.A., Klor, E.F., Miaari, S., & Paserman, M.D. (2009). The Struggle for Palestinian Hearts and Minds: Violence and Public Opinion in the Second Intifada. Boston University Working Paper.

[10] Kavanagh, J.E. (2009). Externally-deadly, but Internally-directed: Constituent Demands, Legitimacy, and the Politics of Violence in the Second Palestinian Intifada. Paper presented at the annual meeting of the Midwest Political Science Association 67th Annual National Conference, The Palmer House Hilton, Chicago, IL.

[11] Abrahms, M. (2004). Are terrorists really rational? The Palestinian example. Orbis, 48, 533-549.

[12] Kydd, H.A., & Walter, B.F. (2002). Sabotaging the Peace: The Politics of Extremist Violence. International Organizations, 56, 263–296.

[13] Pape, R.A. (2003). The Strategic Logic of Suicide Terrorism. American Political Science Review, 97, 1–19.

[14] Zeitzoff, T. (2009). The Microdynamics of Reciprocity in an Asymmetric Conflict: Hamas, Israel, and the 2008-2009 Gaza Conflict. New York University Working Paper.

[15] Kaplan, E. H., Mintz, A., Mishal, S., & Samban, C. (2005). What Happened to Suicide Bombings in Israel? Insights from a Terror Stock Model. Studies in Conflict and Terrorism, 28, 225–235.

[16] Gupta, D.K., & Mundra, K. (2005). Suicide Bombings as a Strategic Weapon: An Empirical Investigation of Hamas and Islamic Jihad. Terrorism and Political Violence, 17, 573–598.

[17] Jeliazkov, I., & Poirier, D. (2008). Dynamic and Structural Features of Intifada Violence: A Markov Process Approach. Bayesian Analysis, 3, 63-78.

[18] Enders, W. & Sandler, T. (1993). The Effectiveness of Anti-Terrorism: A VAR-Intervention Analysis. American Journal of Political Science, 87, 829–44.

[19] Brophy-Baermann, B., & Conybeare, J.A.C. (1994). Retaliating Against Terrorism: Rational Expectations and the Optimality of Rules versus Discretion. American Journal of Political Science, 38, 196–210.

[20] Frisch, H. (2006). Motivation or Capabilities? Israeli Counterterrorism against Palestinian Suicide Bombings and Violence. Journal of Strategic Studies, 29, 843–869.

[21] Ganor, B. (2005). The Counter-Terrorism Puzzle: A Guide for Decision Makers. New Brunswick & London: Transaction Publishers.

[22] Rosendorff, P., & Sandler, T. (2004). Too Much of a Good Thing? The Proactive Response Dilemma. Journal of Conflict Resolution, 48, 657-671.

[23] Rubinstein, D. (2002). Assassinations as a Boomerang. Ha'aretz, March 6, 2002. April 20, 2010.

[24] Siqueira, K., & Sandler, T. (2007). Terrorist versus the Government: Strategic Interaction, Support, and Sponsorship. Journal of Conflict Resolution, 50, 878-898.

[25] Jervis, R. (1976). Perception and misperception in international politics. Princeton University Press Published by Princeton University Press.

[26] Jones, E. E., & Nisbett, R. E. (1971). The actor and the observer: Divergent Perceptions of the Causes of Behavior. New York: General Learning Press.

[27] Kennedy, K. A., & Pronin, E. (2008). When disagreement gets ugly: Perceptions of bias and the escalation of conflict. Personality and Social Psychology Bulletin, 34, 833-848.

[28] Mack, A.J.R. (1975). Why Big Nations Lose Small Wars: The Politics of Asymmetric Conflict. World Politics, 27, 175-200.

[29] Arreguín-Toft, I. (2001). How the Weak Win Wars: A Theory of Asymmetric Conflict. International Security, 26, 93-128.

[30] Arreguín-Toft, I. (2006). How the Weak Win Wars: A Theory of Asymmetric Conflict. Cambridge: Cambridge University Press.

[31] Bueno de Mesquita, E., & Dickson, E.S. (2007). The Propaganda of the Deed: Terrorism, Counterterrorism, and Mobilization. American Journal of Political Science, 51, 364-381.

[32] Bloom, M. (2004). Palestinian Suicide Bombing: Public Support, Market Share, and Outbidding. Political Science Quarterly, 119, 61-88.

[33] Bloom, M. (2005). Dying to Kill: The Allure of Suicide Terror. New York: Columbia University Press.

[34] Berrebi, C., & Klor, E.F. (2006). On Terrorism and Electoral Outcomes: Theory and Evidence from the Israeli–Palestinian Conflict. Journal of Conflict Resolution, 50, 899–925.

[35] Berrebi, C., & Klor, E.F. (2008). Are Voters Sensitive to Terrorism? Direct Evidence from the Israeli Electorate. American Political Science Review, 102, 279–301.

[36] Gordon, C., & Arian, A. (2001). Threat and Decision Making. Journal of Conflict Resolution, 45, 196-215.

[37] Berrebi, C., & Klor, E.F. (2005). The Impact of Terrorism Across Industries: An Empirical Study. CEPR Discussion Paper No. 5360.

[38] Eckstein, Z., & Tsiddon, D. (2004). Macroeconomic Consequences of Terror: Theory and the Case of Israel. Journal of Monetary Economics, 51, 971–1002.

[39] Kydd, A., & Walter, B.F. (2002). Sabotaging the Peace: The Politics of Extremist Violence. International Organization, 56, 263-296.

[40] Ha'aretz (2009). Hamas rockets are bid to sway Israeli election. Ha'aretz.com, April 20, 2010.

[41] Granger, C.W.J. (1969). Investigating Causal Relations by Econometric Models and Cross-Spectral Methods. Econometrica, 37, 424–38.

9.6 Supplementary Information

9.6.1 Data

We obtained data on Israeli and Palestinian fatalities from the human rights organization Bt'selem (www.btselem.org), which is widely respected on both sides of the conflict for providing accurate and reliable data. In addition, we obtained data on Qassam firings from the Israeli Defense Forces (IDF). In this paper we consider three variables. The first is the daily count of Palestinian deaths resulting from Israeli attacks. The remaining two are two different measures of Palestinian aggression against Israel: first, the daily count of Israeli deaths resulting from Palestinian attacks; second, the daily count of Qassam rockets fired from Palestinian into Israeli territory (irrespective of whether anyone died as a result).

Our data cover the period from September 29, 2000 until October 29, 2008 for fatalities, and the period from January 1, 2001 to April 16, 2008 for Qassam firings. All results reported here continue to hold when the fatality data are restricted to the time period available for the Qassams.

We consider the data in two forms: the levels data contains the daily count of Israeli and Palestinian attacks, while the incidence data is a dummy variable which takes the value one on days where at least one attack occurred, and 0 otherwise.

9.6.2 Autoregressive order selection

To test whether Israeli aggression caused Palestinian retaliation and vice-versa, we conducted a series of vector autoregressions (VAR). This technique regresses current Israeli and Palestinian fatalities on previous Israeli and Palestinian fatalities, up to a pre-specified lag.

In a first step, we used an information criterion approach to choose the most appropriate autoregressive order for each VAR process. Lütkepohl (1) showed that Schwarz's Bayesian Information Criterion (SBIC; 2) performed better than Akaike's Information Criterion (AIC; 3-5) in choosing the correct autoregressive order; indeed, SBIC is consistent estimate of the correct lag order (6). We therefore chose the lag structures based on SBIC.

Columns 1-3 in Suppl. Table 9.6.1 reports the SBIC values for the three variables of Israeli and Palestinian aggression. The SBIC values are minimized at lags 5, 4, and 22 for Palestinian fatalities, Israeli fatalities, and Qassam firings, respectively, suggesting that these lags may be the most appropriate ones.

However, note that the difference in information criteria values is small;

under these conditions, a straightforward model choice is not possible (6-7). Therefore we computed Schwarz weights, which have a more intuitive interpretation as the probability of the particular lag order being the optimal one (7). As can be seen in Suppl. Table 9.6.1, columns 4-6, the Schwarz weights for best lags identified above were 0.87, 0.71, and 0.70 for Palestinian fatalities, Israeli fatalities, and Qassam firings, respectively; in addition, the ratios of the probabilities for these lags compared to the next-best lags within each variable were 11.84, 6.99, and 3.45, respectively. Together, these results suggest that the lags identified using SBIC have a high probability of being the optimal ones.

9.6.3 Timeseries order of integration

VAR requires all variables to be of the same order of intergration. We therefore tested for the presence of a unit root using the augmented Dickey-Fuller Test (8). The absence of a unit root implies stationarity of the time series and integration of order 0, and therefore justifies using VAR on the data.

The null hypothesis of the Dickey-Fuller test is the presence of a unit root; a large negative value rejects this hypothesis. We performed the test using the autoregressive orders determined with information criteria, both with and without lag and drift terms. The results are shown in Suppl. Table 9.6.2. The null hypothesis was rejected in all cases, suggesting that the timeseries are stationary and a VAR approach is justified.

9.6.4 Model selection

Next we considered two alternative VAR models, namely ordinary least squares (OLS) and negative binomial (NB) models. The former has been employed in previous papers using this data (9-11); however, from a theoretical point of view negative binomial models are more appropriate in this context since they take into account the count nature of the data. (Note also that negative binomial models are preferable to another group of count data models in this context, namely Poisson models, since the data are overdispersed, cf. Table 9.3.1.)

To choose between these alternative models, a selection criterion approach is not feasible because the two models are too different from each other (6). We therefore used a cross-validation approach: for each of the levels regression models described in the Results section, we omitted a single day from the dataset and fit the model to the remainder of the data. We then generated a prediction for the number of Israeli and Palestinian attacks on the left-out day, and computed the mean squared prediction error:

	SBIC			Schwarz weights		
	(1)	(2)	(3)	(4)	(5)	(6)
	Palestinian	Israeli	Qassam	Palestinian	Israeli	Qassam
Lag	fatalities	fatalities	attacks	fatalities	fatalities	attacks
0	5.35252	3.85969	5.49496	0.0000	0.0000	0.0000
1	5.26646	3.85015	4.95958	0.0000	0.0561	0.0000
2	5.22253	3.85035	4.92645	0.0000	0.0419	0.0000
3	5.20365	3.84974	4.91772	0.0737	0.1022	0.0263
4	5.20465	3.84841*	4.91993	0.0171	0.7146	0.0014
5	5.20196*	3.84994	4.92232	0.8723	0.0763	0.0001
6	5.20419	3.85159	4.91918	0.0334	0.0068	0.0038
7	5.20575	3.85238	4.91861	0.0034	0.0022	0.0081
8	5.20847	3.85487	4.9216	0.0001	0.0001	0.0002
9	5.21045	3.85576	4.92452	0.0000	0.0000	0.0000
10	5.21315	3.85616	4.92483	0.0000	0.0000	0.0000
11	5.2141	3.85753	4.91769	0.0000	0.0000	0.0273
12	5.21671	3.85974	4.91811	0.0000	0.0000	0.0157
13	5.21819	3.85582	4.92079	0.0000	0.0000	0.0005
14	5.21898	3.85749	4.92189	0.0000	0.0000	0.0001
15	5.22135	3.86	4.92484	0.0000	0.0000	0.0000
16	5.22386	3.8627	4.92644	0.0000	0.0000	0.0000
17	5.22658	3.86542	4.9292	0.0000	0.0000	0.0000
18	5.2293	3.86579	4.93207	0.0000	0.0000	0.0000
19	5.2316	3.86837	4.92297	0.0000	0.0000	0.0000
20	5.22733	3.87038	4.92096	0.0000	0.0000	0.0004
21	5.22749	3.86948	4.91617	0.0000	0.0000	0.2023
22	5.23017	3.872	4.91523*	0.0000	0.0000	0.6980
23	5.22568	3.8747	4.91822	0.0000	0.0000	0.0136
24	5.2284	3.87548	4.9197	0.0000	0.0000	0.0019
25	5.22791	3.87171	4.9211	0.0000	0.0000	0.0003
26	5.23005	3.87397	4.92398	0.0000	0.0000	0.0000
27	5.23127	3.87614	4.92629	0.0000	0.0000	0.0000
28	5.23139	3.87709	4.92924	0.0000	0.0000	0.0000
n	2925	2925	2635	2925	2925	2635

Table 9.6.1: Lag order selection statistics. Columns 1-3 report Schwarz's Bayesian Information Criterion (SBIC) values for the three variables of Israeli and Palestinian violence. Columns 4-6 report Schwarz weights for the same variables. See Methods for the description and interpretation of these measures.

Test specification	Test statistic	(1) Palestinian fatalities	(2) Israeli fatalities	(3) Qassam attacks
Basic	Z	-15.1096	-20.4431	-5.1241
	p	0.0000	0.0000	0.0000
With trend	Z	-15.2863	-21.6778	-6.5658
	p	0.0000	0.0000	0.0000
With drift	Z	-15.1096	-20.4431	-5.1241
	p	0.0000	0.0000	0.0000

Table 9.6.2: Dickey-Fuller Test. This table reports the results of the augmented Dickey-Fuller test for the three timeseries of interest. The trend specification includes a trend term in the associated regression, and assumes that the process under the null hypothesis is a random walk (possibly with drift). The drift specifciation assumes that the process under the null hypothesis is a random walk with nonzero drift. Significantly negative test statistics are evidence for stationarity.

The results are shown in Suppl. Table 9.6.3. For all variables, the prediction errors obtained with the OLS model were smaller than those obtained with the NB model. We therefore based our analyses on OLS. (Note that this finding also suggests that the OLS approach used by Jaeger & Paserman (9-10) and Jaeger et al. (11) was probably appropriate.)

9.6.5 Magnitude estimations

To estimate the magnitude of the VAR results, we proceeded as follows. First, the percentage increase in the expected number or probability of attacks on the day after a single attack from the other side was computed by comparing the excess number of attacks on a day after an attack from the other side to the average number of attacks on a given day. For instance, on the first day after a killing of Palestinians by Israelis, an extra 0.08 Qassam rockets are fired, which corresponds to a 6% increase over the 1.37 average daily rockets.

Second, to estimate what percentage of all attacks can be accounted for by retaliation, we computed the number of attacks that were due to retaliation by multiplying the total number of attacks from the respective other side with the excess attacks due to retaliation that resulted from these attacks. For instance, Israeli attacks caused 4478 Palestinian fatalities, each of which led to the firing of an extra 0.08 Qassam rockets, or 358 rockets in total. This corresponds to 10% of the 3645 rockets that were fired altogether. (To obtain a conservative estimate and to avoid bias from different lag structures we restricted ourselves to retaliation on the first day after an attack. In addition, we restricted the estimation period to that for which Qassam data

Model specification	Measure of Palestinian violence	
OLS	Israeli fatalities	Qassam attacks
MSE for prediction of attacks on Israel	2.7132	8.3462
MSE for prediction of attacks on Palestinians	10.6977	12.0430
Negative binomial	Israeli fatalities	Qassam attacks
MSE for prediction of attacks on Israel	3.9297	135700000.0000
MSE for prediction of attacks on Palestinians	3329.1307	5045.0877

Table 9.6.3: Model selection through cross-validation. This table reports the mean squared prediction errors (MSE) obtained from cross-validation. For each of the levels regression models described in the Results section, we omitted a single day from the dataset and fit the model to the remainder of the data. We then generated a prediction for the number of Israeli and Palestinian attacks on the left-out day, and computed the mean squared prediction error. Smaller mean prediction errors are taken as evidence for superiority of the associated model specification.

are available.)

	Levels				Incidence			
	DV: Attacks on Israel		DV: Attacks on Palestinians		DV: Attacks on Israel		DV: Attacks on Palestinians	
Measure of Palestinian aggression	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities
L1is	0.446	0.092	0.027	0.156	0.172	0.082	0.018	0.099
	[0.000]***	[0.005]***	[0.549]	[0.056]*	[0.000]***	[0.000]***	[0.494]	[0.000]***
L2is	0.134	0.032	0.057	0.08	0.113	0.068	0.005	0.044
	[0.044]**	[0.160]	[0.463]	[0.113]	[0.000]***	[0.003]***	[0.842]	[0.108]
L3is	0.121	0.041	0.098	0.106	0.111	0.062	-0.024	0.051
	[0.069]*	[0.054]*	[0.327]	[0.025]**	[0.000]***	[0.006]***	[0.370]	[0.065]*
L4is	-0.059	0.054	-0.119	0.077	0.051	0.048	0.017	0.083
	[0.276]	[0.232]	[0.137]	[0.396]	[0.032]**	[0.027]**	[0.524]	[0.002]***
L5is	-0.021		-0.041		0.069		-0.009	
	[0.586]		[0.239]		[0.004]***		[0.739]	
L6is	0.051		-0.021		0.04		-0.011	
	[0.211]		[0.518]		[0.091]*		[0.676]	
L7is	0.047		0.008		0.075		0.048	
	[0.228]		[0.788]		[0.002]***		[0.075]*	
L8is	-0.03		-0.007		0.014		-0.056	
	[0.490]		[0.815]		[0.550]		[0.036]**	
L9is	-0.04		-0.026		0.006		0.026	
	[0.305]		[0.330]		[0.800]		[0.342]	
L10is	-0.007		0.028		0.044		0.013	
	[0.834]		[0.261]		[0.061]*		[0.633]	
L11is	0.063		-0.016		0.035		0.022	
	[0.130]		[0.525]		[0.141]		[0.421]	
L12is	0.035		0.027		0.028		-0.008	
	[0.532]		[0.356]		[0.226]		[0.767]	
L13is	-0.002		-0.041		0.011		0	
	[0.974]		[0.084]*		[0.648]		[0.990]	
L14is	0.05		0.097		0.055		-0.023	
	[0.323]		[0.008]***		[0.022]**		[0.389]	
L15is	0.005		-0.073		-0.012		0.047	
	[0.914]		[0.102]		[0.603]		[0.081]*	
L16is	-0.052		0.018		0.035		-0.061	
	[0.197]		[0.521]		[0.123]		[0.024]**	
L17is	-0.005		-0.051		-0.016		-0.043	
	[0.902]		[0.021]**		[0.468]		[0.111]	
L18is	-0.057		-0.009		0.012		-0.023	
	[0.092]*		[0.676]		[0.603]		[0.379]	
L19is	0.057		0.03		0.026		0.039	
	[0.410]		[0.382]		[0.252]		[0.142]	
L20is	0.017		0.04		0.019		-0.012	
	[0.750]		[0.333]		[0.413]		[0.642]	
L21is	0.054		-0.036		0.008		-0.009	
	[0.207]		[0.345]		[0.727]		[0.727]	
L22is	0.067		0.053		0.021		0.025	
	[0.215]		[0.425]		[0.341]		[0.339]	
L1pa	0.08	0.011	0.183	0.172	0.038	0.012	0.129	0.124
	[0.001]***	[0.332]	[0.000]***	[0.000]***	[0.010]***	[0.343]	[0.000]***	[0.000]***
L2pa	0.013	0.01	0.146	0.153	0.01	0.028	0.107	0.103
	[0.595]	[0.483]	[0.000]***	[0.001]***	[0.516]	[0.020]**	[0.000]***	[0.000]***
L3pa	-0.041	0.005	0.109	0.115	0.005	0.027	0.045	0.039
	[0.048]**	[0.612]	[0.247]	[0.151]	[0.739]	[0.026]**	[0.025]**	[0.041]**
L4pa	-0.02	-0.004	0.038	0.025	-0.01	0.003	0.103	0.088
	[0.353]	[0.634]	[0.254]	[0.436]	[0.494]	[0.799]	[0.000]***	[0.000]***
L5pa	-0.007	0.043	0.091	0.069	-0.03	0.051	0.096	0.099
	[0.666]	[0.086]*	[0.003]***	[0.019]**	[0.037]**	[0.000]***	[0.000]***	[0.000]***
Constant	0.136	0.173	0.679	0.611	0.025	0.026	0.27	0.239
	[0.055]*	[0.000]***	[0.000]***	[0.000]***	[0.101]	[0.005]***	[0.000]***	[0.000]***
Observations	2641	2948	2641	2948	2641	2948	2641	2948
R-squared	0.481	0.033	0.175	0.164	0.434	0.048	0.088	0.102

Table 9.6.4: Full regression table for basic specification. All regressions are OLS models and are estimated using heteroskedasticity-consistent standard errors. Robust p values are shown in brackets. We denote significance at 10% with *, significance at 5% with **, and significance at 1% with ***. The dependent variable are attacks by Palestinians on Israel (columns 1 and 2), and attacks by Israel on Palestinians (columns 3 and 4). The right-hand side variables are the number or incidence of Israeli and Palestinian attacks at lags ranging from 4 to 22 (see Methods for choice of lag structure). Lagged variables are expressed as L#is and L#pa, respectively, where # is the lag order. For both the dependent and independent variables, columns 1 and 3 use Qassam firings as a measure of Palestinian aggression, while columns 2 and 4 use Israeli fatalities as a measure of Palestinian aggression.

	Levels				Incidence			
	DV: Attacks on Israel		DV: Attacks on Palestinians		DV: Attacks on Israel		DV: Attacks on Palestinians	
Measure of Palestinian aggression	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities
pa	0.168	0.032	0.25	0.122	0.031	0.068	0.054	0.153
	[0.002]***	[0.001]***	[0.004]***	[0.006]***	[0.040]**	[0.000]***	[0.040]**	[0.000]***
L1is	0.442	0.087	-0.084	0.145	0.171	0.075	0.009	0.087
	[0.000]***	[0.008]***	[0.185]	[0.077]*	[0.000]***	[0.001]***	[0.747]	[0.001]***
L2is	0.124	0.03	0.023	0.076	0.112	0.065	-0.001	0.034
	[0.030]**	[0.179]	[0.689]	[0.121]	[0.000]***	[0.004]***	[0.974]	[0.215]
L3is	0.104	0.038	0.068	0.101	0.111	0.059	-0.03	0.041
	[0.040]**	[0.072]*	[0.390]	[0.033]**	[0.000]***	[0.008]***	[0.264]	[0.132]
L4is	-0.039	0.051	-0.104	0.071	0.05	0.043	0.014	0.076
	[0.351]	[0.251]	[0.116]	[0.431]	[0.033]**	[0.050]**	[0.594]	[0.005]***
L5is	-0.014		-0.036		0.069		-0.013	
	[0.699]		[0.254]		[0.004]***		[0.637]	
L6is	0.055		-0.034		0.04		-0.013	
	[0.162]		[0.248]		[0.088]*		[0.618]	
L7is	0.045		-0.004		0.074		0.044	
	[0.229]		[0.892]		[0.002]***		[0.104]	
L8is	-0.028		0		0.016		-0.057	
	[0.498]		[0.998]		[0.502]		[0.033]**	
L9is	-0.035		-0.016		0.005		0.025	
	[0.348]		[0.511]		[0.826]		[0.348]	
L10is	-0.012		0.03		0.044		0.01	
	[0.714]		[0.197]		[0.063]*		[0.700]	
L11is	0.066		-0.032		0.035		0.02	
	[0.108]		[0.229]		[0.149]		[0.464]	
L12is	0.031		0.018		0.028		-0.009	
	[0.581]		[0.523]		[0.222]		[0.725]	
L13is	0.005		-0.04		0.011		-0.001	
	[0.924]		[0.096]*		[0.648]		[0.972]	
L14is	0.033		0.085		0.055		-0.026	
	[0.489]		[0.009]***		[0.020]**		[0.330]	
L15is	0.018		-0.075		-0.013		0.048	
	[0.704]		[0.080]*		[0.561]		[0.078]*	
L16is	-0.054		0.031		0.037		-0.063	
	[0.170]		[0.314]		[0.105]		[0.020]**	
L17is	0.004		-0.05		-0.015		-0.042	
	[0.928]		[0.027]**		[0.505]		[0.118]	
L18is	-0.055		0.005		0.013		-0.024	
	[0.088]*		[0.827]		[0.581]		[0.365]	
L19is	0.052		0.016		0.025		0.038	
	[0.438]		[0.627]		[0.275]		[0.157]	
L20is	0.01		0.036		0.02		-0.013	
	[0.835]		[0.306]		[0.404]		[0.614]	
L21is	0.06		-0.049		0.008		-0.01	
	[0.140]		[0.188]		[0.718]		[0.715]	
L22is	0.058		0.036		0.02		0.024	
	[0.202]		[0.488]		[0.358]		[0.361]	
L1pa	0.049	0.005	0.163	0.17	0.034	0.003	0.127	0.122
	[0.073]*	[0.638]	[0.000]***	[0.000]***	[0.021]**	[0.792]	[0.000]***	[0.000]***
L2pa	-0.012	0.005	0.143	0.152	0.006	0.021	0.107	0.098
	[0.565]	[0.720]	[0.000]***	[0.001]***	[0.674]	[0.081]*	[0.000]***	[0.000]***
L3pa	-0.06	0.001	0.119	0.115	0.004	0.025	0.044	0.035
	[0.013]**	[0.877]	[0.199]	[0.150]	[0.811]	[0.043]**	[0.026]**	[0.067]*
L4pa	-0.026	-0.005	0.042	0.025	-0.013	-0.003	0.104	0.088
	[0.220]	[0.578]	[0.181]	[0.428]	[0.371]	[0.823]	[0.000]***	[0.000]***
L5pa	-0.022	0.041	0.093	0.064	-0.033	0.045	0.098	0.091
	[0.175]	[0.108]	[0.002]***	[0.037]**	[0.024]**	[0.000]***	[0.000]***	[0.000]***
Constant	0.021	0.154	0.645	0.589	0.016	0.01	0.269	0.235
	[0.803]	[0.000]***	[0.000]***	[0.000]***	[0.283]	[0.268]	[0.000]***	[0.000]***
Observations	2641	2948	2641	2948	2641	2948	2641	2948
R-squared	0.503	0.037	0.21	0.167	0.435	0.058	0.089	0.112

Table 9.6.5: Full regression table, controlling for same-day events. All regressions are as described for Table 9.6.4, except a control variable is added for attacks taking place on the same day.

	Levels				Incidence			
	DV: Attacks on Israel		DV: Attacks on Palestinians		DV: Attacks on Israel		DV: Attacks on Palestinians	
Measure of Palestinian aggression	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities
L1is	0.162	0.013	0.048	0.087	0.147	0.066	0.039	0.154
	[0.003]***	[0.407]	[0.127]	[0.097]*	[0.000]***	[0.079]*	[0.263]	[0.002]***
L2is	0.123	0.014	0.005	0.014	0.113	0.061	0.008	0.03
	[0.009]***	[0.385]	[0.877]	[0.698]	[0.000]***	[0.012]**	[0.784]	[0.339]
L3is	0.072	0.045	-0.026	0.082	0.13	0.041	-0.041	0.04
	[0.031]**	[0.056]*	[0.535]	[0.064]*	[0.000]***	[0.077]*	[0.170]	[0.189]
L4is	0.025	-0.004	0.008	-0.047	0.045	0.031	0.002	0.082
	[0.434]	[0.687]	[0.767]	[0.081]*	[0.087]*	[0.165]	[0.938]	[0.006]***
L5is	-0.008		-0.058		0.07		-0.005	
	[0.786]		[0.038]**		[0.008]***		[0.880]	
L6is	0.099		0.016		0.006		-0.039	
	[0.076]*		[0.600]		[0.811]		[0.202]	
L7is	-0.031		0		0.088		0.052	
	[0.374]		[0.993]		[0.001]***		[0.094]*	
L8is	0.055		-0.03		0.03		-0.063	
	[0.283]		[0.226]		[0.255]		[0.038]**	
L9is	-0.012		-0.023		0.023		0.04	
	[0.770]		[0.337]		[0.395]		[0.186]	
L10is	-0.044		0.025		0.043		0.024	
	[0.225]		[0.274]		[0.096]*		[0.427]	
L11is	0.046		0.025		0.048		0.029	
	[0.169]		[0.164]		[0.077]*		[0.342]	
L12is	0.045		-0.011		0.008		0	
	[0.426]		[0.596]		[0.759]		[0.997]	
L13is	-0.011		-0.027		0.031		-0.01	
	[0.863]		[0.206]		[0.243]		[0.754]	
L14is	0.033		0.025		0.056		-0.023	
	[0.518]		[0.275]		[0.034]**		[0.448]	
L15is	0.108		0.001		-0.021		0.039	
	[0.065]*		[0.965]		[0.408]		[0.204]	
L16is	-0.074		0.008		0.022		-0.056	
	[0.138]		[0.798]		[0.368]		[0.066]*	
L17is	0.012		-0.001		-0.015		-0.049	
	[0.672]		[0.958]		[0.562]		[0.105]	
L18is	0.003		-0.021		0.007		-0.024	
	[0.906]		[0.355]		[0.797]		[0.424]	
L19is	-0.016		-0.007		0.043		0.044	
	[0.525]		[0.760]		[0.103]		[0.149]	
L20is	0.036		0.04		0.028		-0.018	
	[0.429]		[0.214]		[0.283]		[0.544]	
L21is	0.047		0.035		-0.001		-0.02	
	[0.160]		[0.395]		[0.951]		[0.507]	
L22is	0.002		-0.047		0.023		0.03	
	[0.950]		[0.052]*		[0.363]		[0.313]	
L1pa	0.007	0.002	0.198	0.167	0.033	0.012	0.128	0.134
	[0.470]	[0.874]	[0.000]***	[0.000]***	[0.048]**	[0.320]	[0.000]***	[0.000]***
L2pa	0.017	0	0.114	0.168	0.014	0.025	0.114	0.095
	[0.126]	[0.980]	[0.000]***	[0.006]***	[0.358]	[0.040]**	[0.000]***	[0.000]***
L3pa	-0.013	0.005	0.187	0.028	0.004	0.024	0.035	0.039
	[0.291]	[0.556]	[0.171]	[0.368]	[0.784]	[0.057]*	[0.116]	[0.048]**
L4pa	-0.01	0.004	0.036	0.049	-0.008	0.004	0.097	0.082
	[0.507]	[0.593]	[0.414]	[0.108]	[0.591]	[0.752]	[0.000]***	[0.000]***
L5pa	-0.01	0.027	0.073	0.104	-0.042	0.05	0.104	0.102
	[0.518]	[0.236]	[0.017]**	[0.003]***	[0.006]***	[0.000]***	[0.000]***	[0.000]***
Constant	0.235	0.228	0.678	0.683	0.025	0.034	0.279	0.242
	[0.000]***	[0.000]***	[0.000]***	[0.000]***	[0.131]	[0.000]***	[0.000]***	[0.000]***
Observations	2157	2702	2157	2702	2157	2702	2157	2702
R-squared	0.283	0.007	0.169	0.121	0.391	0.028	0.088	0.085

Table 9.6.6: Full regression table, omitting mutual events at t-1. All regressions are as described for Table 9.6.4, except that events at t-1 are omitted from the analysis.

	Levels				Incidence			
	DV: Attacks on Israel		DV: Attacks on Palestinians		DV: Attacks on Israel		DV: Attacks on Palestinians	
Measure of Palestinian aggression	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities	Qassam attacks	Israeli fatalities
L1is	0.427	0.064	0.034	0.152	0.148	0.04	0.042	0.078
	[0.000]***	[0.051]*	[0.453]	[0.066]*	[0.000]***	[0.080]*	[0.112]	[0.005]***
L2is	0.121	0.006	0.061	0.077	0.092	0.028	0.027	0.026
	[0.072]*	[0.810]	[0.433]	[0.126]	[0.000]***	[0.221]	[0.302]	[0.358]
L3is	0.109	0.015	0.104	0.103	0.092	0.022	-0.004	0.031
	[0.106]	[0.460]	[0.304]	[0.030]**	[0.000]***	[0.322]	[0.865]	[0.265]
L4is	-0.07	0.026	-0.113	0.075	0.035	0.008	0.034	0.064
	[0.200]	[0.571]	[0.151]	[0.412]	[0.136]	[0.717]	[0.204]	[0.019]**
L5is	-0.03		-0.037		0.055		0.005	
	[0.439]		[0.293]		[0.021]**		[0.857]	
L6is	0.043		-0.02		0.028		-0.001	
	[0.283]		[0.544]		[0.227]		[0.977]	
L7is	0.039		0.009		0.064		0.057	
	[0.318]		[0.763]		[0.007]***		[0.032]**	
L8is	-0.036		-0.007		0.005		-0.048	
	[0.387]		[0.833]		[0.839]		[0.068]*	
L9is	-0.046		-0.027		-0.004		0.034	
	[0.238]		[0.308]		[0.881]		[0.206]	
L10is	-0.014		0.028		0.034		0.021	
	[0.670]		[0.272]		[0.143]		[0.423]	
L11is	0.055		-0.016		0.026		0.031	
	[0.180]		[0.532]		[0.278]		[0.245]	
L12is	0.026		0.027		0.017		0.003	
	[0.649]		[0.361]		[0.442]		[0.924]	
L13is	-0.01		-0.04		0.001		0.009	
	[0.856]		[0.086]*		[0.963]		[0.738]	
L14is	0.042		0.097		0.044		-0.012	
	[0.404]		[0.009]***		[0.063]*		[0.649]	
L15is	-0.001		-0.072		-0.021		0.057	
	[0.982]		[0.105]		[0.358]		[0.032]**	
L16is	-0.058		0.017		0.024		-0.049	
	[0.143]		[0.524]		[0.288]		[0.068]*	
L17is	-0.014		-0.051		-0.027		-0.034	
	[0.731]		[0.021]**		[0.231]		[0.194]	
L18is	-0.064		-0.009		0		-0.014	
	[0.059]*		[0.682]		[0.994]		[0.587]	
L19is	0.05		0.031		0.015		0.047	
	[0.465]		[0.373]		[0.517]		[0.072]*	
L20is	0.007		0.04		0.006		0	
	[0.898]		[0.331]		[0.802]		[0.986]	
L21is	0.041		-0.034		-0.006		0.002	
	[0.319]		[0.344]		[0.783]		[0.937]	
L22is	0.05		0.054		0.004		0.04	
	[0.359]		[0.425]		[0.863]		[0.126]	
L1pa	0.086	0.01	0.169	0.165	0.05	0.003	0.09	0.1
	[0.001]***	[0.362]	[0.000]***	[0.000]***	[0.001]***	[0.831]	[0.000]***	[0.000]***
L2pa	0.02	0.009	0.134	0.147	0.023	0.019	0.068	0.08
	[0.392]	[0.534]	[0.000]***	[0.002]***	[0.120]	[0.118]	[0.001]***	[0.000]***
L3pa	-0.034	0.003	0.097	0.109	0.018	0.019	0.007	0.017
	[0.102]	[0.731]	[0.307]	[0.175]	[0.208]	[0.121]	[0.713]	[0.358]
L4pa	-0.012	-0.007	0.026	0.018	0.004	-0.007	0.064	0.065
	[0.567]	[0.409]	[0.442]	[0.553]	[0.766]	[0.595]	[0.001]***	[0.001]***
L5pa	0	0.039	0.078	0.061	-0.015	0.039	0.056	0.074
	[0.985]	[0.127]	[0.011]**	[0.034]**	[0.296]	[0.002]***	[0.005]***	[0.000]***
year==2002	-0.066	0.659	0.729	-0.145	0.005	0.048	0.12	-0.015
	[0.164]	[0.000]***	[0.001]***	[0.675]	[0.726]	[0.350]	[0.001]***	[0.764]
year==2003	0.184	0.154	0.083	-0.496	0.071	-0.075	-0.032	-0.097
	[0.096]*	[0.324]	[0.638]	[0.120]	[0.001]***	[0.127]	[0.403]	[0.069]*
year==2004	0.201	-0.068	0.419	-0.065	0.124	-0.114	-0.018	-0.042
	[0.017]**	[0.562]	[0.057]*	[0.847]	[0.000]***	[0.017]**	[0.671]	[0.426]
year==2005	0.204	-0.116	-0.461	-0.898	0.086	-0.124	-0.234	-0.264
	[0.071]*	[0.304]	[0.004]***	[0.008]***	[0.000]***	[0.011]**	[0.000]***	[0.000]***
year==2006	0.923	-0.251	0.023	-0.21	0.263	-0.165	-0.189	-0.11
	[0.000]***	[0.012]**	[0.938]	[0.555]	[0.000]***	[0.000]***	[0.000]***	[0.044]**
year==2007	0.971	-0.231	-0.354	-0.586	0.283	-0.159	-0.296	-0.199
	[0.000]***	[0.026]**	[0.249]	[0.079]*	[0.000]***	[0.001]***	[0.000]***	[0.000]***
year==2008	2.599	-0.197	0.283	-0.416	0.311	-0.143	-0.208	-0.204
	[0.006]***	[0.067]*	[0.662]	[0.328]	[0.000]***	[0.003]***	[0.005]***	[0.000]***
Constant	-0.077	0.208	0.666	1.092	-0.04	0.159	0.375	0.433
	[0.060]*	[0.071]*	[0.000]***	[0.002]***	[0.011]**	[0.001]***	[0.000]***	[0.000]***
Observations	2641	2948	2641	2948	2641	2948	2641	2948
R-squared	0.49	0.059	0.185	0.169	0.447	0.086	0.12	0.122

Table 9.6.7: Full regression table, including year dummies. All regressions are as described for Table 9.6.4, except that the specfications reported here include year dummies to allow for time trends.

Bibliography

[1] Lütkepohl, H. (1985). Comparison of Criteria for estimating the Order of a Vector Autoregressive Process. Journal of Time Series Analysis, 6, 35-36, 47, 50.

[2] Schwarz, G.E. (1978). Estimating the dimension of a model. Annals of Statistics, 6, 461–464.

[3] Akaike, H. (1973). Information theory and an extension of the maximum likelihood principle. In B. N. Petrov & F. Caski (Eds.), Proceedings of the Second International Symposium on Information Theory. Budapest: Akademiai Kiado.

[4] Akaike, H. (1974). A new look at the statistical model identification. IEEE Transactions on Automatic Control, 19, 716-723.

[5] Akaike, H. (1978). On the likelihood of a time series model. The Statistician, 27, 217-235.

[6] Burnham, K.P., & Anderson, D.R. (2002). Model selection and multimodel inference: a practical information-theoretic approach. 2nd Edition. New York, NY: Springer-Verlag.

[7] Wagenmakers, E.J., & Farrell, S. (2004). AIC model selection using Akaike weights. Psychonomic Bulletin & Review, 11, 192-196.

[8] Elliott, G., Rothenberg, T.J., & Stock, J.H. (1996). Efficient Tests for an Autoregressive Unit Root. Econometrica, 64, 813–836.

[9] Jaeger, D.A., & Paserman, M.D. (2006). Israel, the Palestinian Factions, and the Cycle of Violence. American Economic Review, 96, 45–49.

[10] Jaeger, D.A., & Paserman, M.D. (2008). The Cycle of Violence? An Empirical Analysis of Fatalities in the Palestinian-Israeli Conflict. American Economic Review, 98, 1591–1604.

[11] Jaeger, D.A., Klor, E.F., Miaari, S., & Paserman, M.D. (2009). The Struggle for Palestinian Hearts and Minds: Violence and Public Opinion in the Second Intifada. Boston University Working Paper.

Chapter 10

The lifespans of winners and runners-up in U.S. presidential elections do not differ

10.1 Introduction

The effect of holding a high-stress office such as that of U.S. President has been suggested to affect health outcomes such as expected lifespan. However, in seeming opposition to this popular belief, it has recently been shown that U.S. Presidents in fact live longer than average males of the same birth cohort, conditional on having survived until the age of first inauguration (Olshansky, 2011). However, comparing the lifespans of U.S. Presidents to average males of the same birth cohort neglects the fact that presidents are highly untypical: they are richer, better educated, and more urbanized than the typical male. These factors typically increase lifespan, and thus comparing presidents to average males is likely to result in an underestimate of the effect of holding office on lifespan. Here we control for this selection by comparing the winners and runner-ups of U.S. presidential elections from 1792 until the present. These groups are similar in the socioeconomic variables mentioned above, but differ in whether they have served as president. We find that winning an election, the number of elections won or participated in, the success rate in winning elections, the number of days spent in office, or ever serving as president, do not affect expected lifespan compared to runners-up. We observe a weak positive effect of survived assassination attempts on lifespan. Together, these results suggest that presidents' lifespan is not affected by holding office.

10.2 Methods

We obtained the birth and death dates of all U.S. presidents and the runners-up in the electoral college from 1792 until present (variables *dob* and *dob* in Table 1, respectively), and used these dates to calculate the lifespan of each president and runner-up (*dayslived*). We further computed the number of days spent in office (*daysoffice*), the number of elections won (*numwins*), the number of elections participated in (*numelect*), an indicator variable for whether a candidate ever won an election (*everwon*), the success rate at winning elections, defined as the ratio of number of elections won to number of elections participated in (*successrate*); further, an indicator variable for whether a candidate ever served as president (*everpres*; note that this variable differs from everwon since some vice-presidents took office after the death of the president and thus were not elected), and an indicator variable for whether a candidate was ever the victim of an unsuccessful assassination attempt (*assass_attempt*). Presidents who were victims of successful assassination attempts were excluded from the analysis, as were presidents who are still alive today and for whom lifespan can therefore not be calculated. We then compared the average lifespan of presidents and runners-up, and assessed the significance of any differences using OLS regressions, where daysalive was the dependent variable and the election outcome variables described above were explanatory variables. Date of birth was always included as a control variable to account for general changes in life expectancy over time.

10.3 Results

Figure 10.3.1 presents a scatterplot comparing the lifespan of runners-up to that of the winners of U.S. presidential elections. The diagonal line represents equal lifespans for the runner-up and the winner; data points below the line indicate that the runner-up lived longer than the winner, while data points above the line indicate the opposite relationship. Of the 50 elections in which neither the winner nor the runner-up were later assassinated, we found that the runner-up outlived the winner in 29 cases, while the winner lived longer in the remaining 21 cases. A binomial test of this observed proportion against a theoretically predicted proportion of 0.5 resulted in a result of $P = 0.3222$ (two-sided binomial test), suggesting that winners of presidential elections live somewhat less long than runners-up, but not significantly so. This result was confirmed by a series of OLS regressions, with daysalive as the dependent variable and daysoffice, numwins, numelect, everwon, successrate,

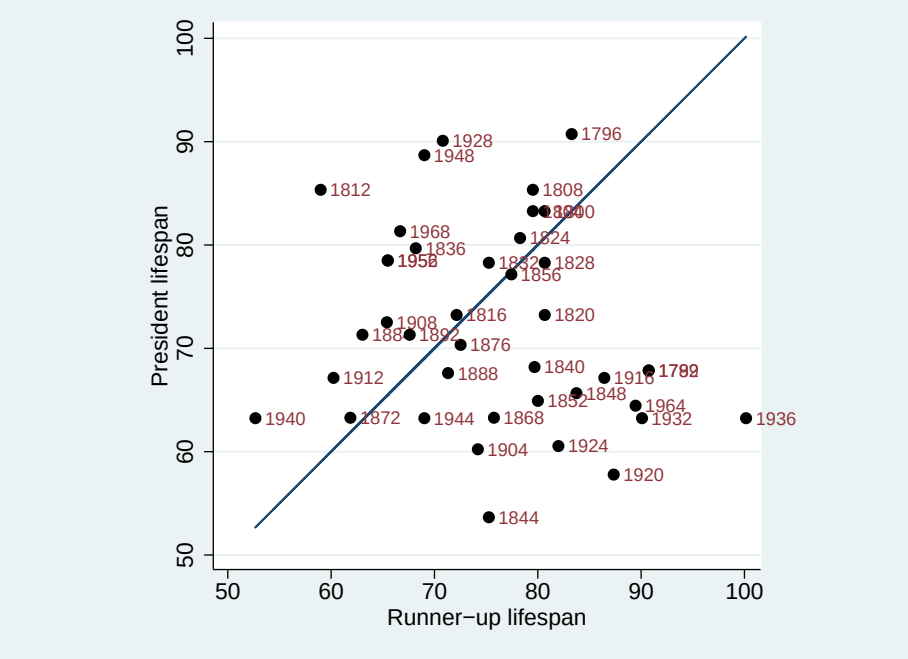

Figure 10.3.1: Relationship between lifespan of runner-up (x-axis) and winner (y-axis) of each U.S. presidential election from 1792 until present. The 45 degree line represents equal lifespans for winner and runner-up; data points above this line indicate that the winner lived longer than the runner-up, data points below the line indicate the opposite. The labels on the data points indicate the year of the election. Data for presidents who were assassinated or are still alive is not shown.

everpres, assass_attempt, and dob as independent variables. The results are shown in Table 10.3.1: we found that none of the election outcome variables significantly predicted lifespan. Surprisingly, the effect of unsuccessful assassination attempts on lifespan was positive and near significance at the $P = 0.05$ level; however, this result is potentially confounded by the possibility that healthier presidents may be more likely to survive assassination attempts. Together, these results suggest that winners of U.S. presidential elections do not live longer or shorter than runners-up.

10.4 Discussion

The purpose of this study was to assess the effect of winning an election and serving as U.S. President on lifespan. Popular belief maintains that holding the stressful office of U.S. President should decrease lifespan, while a recent

VARIABLES	(1) model1 daysalive	(2) model2 daysalive	(3) model3 daysalive	(4) model4 daysalive	(5) model5 daysalive	(6) model6 daysalive	(7) model7 daysalive Presidents only	(8) model8 daysalive
Daysoffice	-0.152							-0.687
	(0.522)							(2.403)
Numwins		251.1						1,197
		(841.4)						(2,774)
Numelect		-1,099	-933.7					-1,827
		(1,033)	(818.3)					(1,192)
Everwon			194.5					-1,488
			(1,513)					(5,475)
Successrate				-581.7				-1,722
				(1,511)				(6,217)
Everpres					-33.63			2,373
					(1,333)			(3,066)
assass_attempt						2,983	4,359	4,121*
						(2,219)	(2,703)	(2,384)
Dob	0.125**	0.127**	0.127**	0.117**	0.123**	0.0869	0.0508	0.0821
	(0.0531)	(0.0572)	(0.0574)	(0.0546)	(0.0519)	(0.0568)	(0.0820)	(0.0681)
Constant	31,025***	32,499***	32,338***	30,923***	30,815***	28,951***	26,667***	31,474***
	(2,503)	(3,228)	(3,164)	(2,501)	(2,458)	(2,563)	(3,470)	(4,325)
Observations	40	38	38	38	40	40	20	38
R-squared	0.137	0.141	0.140	0.121	0.135	0.192	0.362	0.258

Table 10.3.1: Effect of Election Outcomes on Lifespan. Each column shows the effect of a particular election outcome variable on the lifespan of the winners and runners-up in all U.S. presidential elections since 1792. Presidents who were assassinated (Lincoln, Garfield, McKinley, Kennedy) are omitted, as are presidents who are still alive as of December 2011. Robust standard errors in parentheses. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

study has suggested that presidents may live longer than average males. We control for the selection bias which afflicts a straight comparison of presidents to the average population by instead comparing winners to runners-up in U.S. presidential elections. These two groups differ largely in terms of whether they ever held office as U.S. president, and can thus be compared to identify the effect of holding the office of president on lifespan. This analysis reveals that, while election winners tend to die earlier than runners-up, holding office has no statistically significant effect on lifespan. Thus, the stress associated with holding the office of U.S. President is either not severe enough to result in detectable differences in lifespan, or it is outweighed by a "winner effect" (Redelmeier & Singh, 2001; Rablen & Oswald, 2008; Sylvestre et al., 2006; Han et al., 2011).

Bibliography

[1] Olshansky, J.S. (2011). Aging of US Presidents. JAMA 306(23), 2537-2633.

[2] Redelmeier, D. A. and Singh, S. M. (2001). Survival in Academy Award—winning actors and actresses. Ann. Intern. Med. 134 955–962.

[3] Rablen, M. D. and Oswald, A. J. (2008). Mortality and immortality: The Nobel Prize as an experiment into the effect of status upon longevity. Journal of Health Economics 27 1462–1471.

[4] Sylvestre, M. P., Huszti, E. and Hanley, J. A. (2006). Do Oscar winners live longer than less successful peers? A reanalysis of the evidence. Ann. Intern. Med. 145 361–363.

[5] Han, X., Small, D. S., Foster, D.P., Patel, V. (2011). The effect of winning an Oscar Award on survival: Correcting for healthy performer survivor bias with a rank preserving structural accelerated failure time model. Ann. Appl. Stat. 5(2A), 746-772.

Chapter 11

Certain Outcomes vs. Lotteries for Incentive Design

11.1 Summary

Numerous experiments in th behavioral and social sciences, both in the lab and in the field, incentivize participation by offering monetary rewards. Here I use insights from behavioral economics about risk aversion and probability weighting to make recommendations for the optimal design of such incentive schemes. I show that, for individual participants and given a fixed budget, certain payments for participation are superior to coinflip lotteries. In contrast, across participants it can be advantageous to offer lotteries in which one participant receives a large reward for participation, and the remaining participants receive nothing, compared to a scenario where each participant gets a small fee.

11.2 Setup

This note asks how to optimally incentivize participation in a research study with either certain payments or lotteries. Define a prospect as the utility of a lottery, where a lottery is a discrete distribtion of probabilities over a number of outcomes. In evaluating prospects, people take into account both the amounts and the probablilities involved; to the best of current knowledge, the value of a two-outcome prospect with outcomes x and y can be described by this function (Kahneman & Tversky, 1979; Prelec, 1998):

$$V = \pi(p)\, v(x) + (1 - \pi(p))\, v(y)$$

where $|x| > |y|$, $v(\cdot)$ is the utility of an outcome, and $\pi(p)$ is the subjective probability of that outcome as a function of its true probability p.

An alternative way of writing this value is

$$V = v(y) + \pi(p)(v(x) - v(y)).$$

For a certain payment, this reduces to

$$V = v(y).$$

The commonly used functions to model how people actually evaluate prospects are as follows:

$$v(x) = x^{\sigma}$$
$$\pi(p) = \frac{1}{\exp\left(\left(\ln \frac{1}{p}\right)^{\alpha}\right)}.$$

The parameter σ reflects risk aversion (as captured by the concavity of the value function), and the parameter α reflects non-linear (s-shaped) probability weighting.

11.3 Example

Let's work through an example: is it better to offer people a certain payment of PKR 100, or a lottery where they get \$200 with $p = 0.5$, or \$0 with $p = 0.5$?

For the lottery to be superior to the certain outcome, the following needs to be true:

$$v(100) < v(0) + \pi(0.5)(v(200) - v(0))$$

Now substitute the functional forms:

$$100^{\sigma} < 0^{\sigma} + \frac{1}{\exp\left(\left(\ln \frac{1}{0.5}\right)^{\alpha}\right)}(200^{\sigma} - 0^{\sigma}).$$

To make *a priori* judgments about whether to offer people the lottery or the certain outcome, we need estimates for the parameters α and σ. The best estimates from developing countries that I'm aware of are cited in Tanaka et al. (2010): they estimate σ at 0.59, 0.63, and 0.48 in southern Vietnam, northern Vietnam, and rural China, respectively (the China data come from Elaine Liu), and α at 0.74, 0.74, and 0.69, respectively. Let's take the average of these and use

$$\sigma = 0.57$$

$$\alpha = 0.72.$$

Then we obtain:

$$\begin{aligned} 100^{.57} &< 0^{.57} + \frac{1}{\exp\left((\ln 2)^{0.72}\right)}\left(200^{0.57} - 0^{0.57}\right) \\ 13.80 &< 0 + 0.46\,(20.49 - 0) \\ 13.80 &< 9.51 \end{aligned}$$

This condition is not fulfilled; thus the certain outcome in this case would be considered superior to the lottery.

11.4 Extensions

11.4.1 A decision rule for coinflip lotteries

Let's say we want to decide whether to incentivize people with a certain outcome or a two-outcome lottery, we have a fixed total budget B for each participant, and for the sake of simplicity we want to use a coinflip lottery, i.e. $p = 0.5$. We are free to decide the two possible outcomes of the lottery, x and y. Because the expected cost of the lottery must equal the budget, the budget constraint is:

$$px + (1 - p)\, y = B$$

Since we are dealing with a coinflip lottery, this simplifies to:

$$x + y = 2B.$$

The value of the lottery is therefore:

$$\begin{aligned} V &= \pi\,(0.5)\, v\,(x) + \pi\,(0.5)\, v\,(2B - x) \\ &= \pi\,(0.5)\, x^{\sigma} + \pi\,(0.5)\,(2B - x)^{\sigma}. \end{aligned}$$

Maximizing this expression with respect to x yields:

$$\begin{aligned} \frac{\partial V}{\partial x} &= \pi\,(0.5)\,\sigma x^{\sigma-1} - \pi\,(0.5)\,\sigma\,(2B - x)^{\sigma-1} \\ x &= B. \end{aligned}$$

In other words, the value of a coinflip lottery is always lower than the value of the safe outcome with the same expected cost to the experimenter, up to the point where the two outcomes of the lottery are identical and equal to B; in this case the lottery is just as valuable as the safe outcome. Together, this means that coinflip lotteries are never superior to safe outcomes when incentivizing indvidual participants.

11.4.2 A decision rule for paying each participant a fixed participation fee vs. entering them in a draw

Now let's assume we have a fixed budget B which we can either divide up among all N participants, so that each participant gets $\frac{B}{N}$, or we can play a lottery in which one person gets B while all others get 0. The lottery is superior to the fixed payment if

$$\left(\frac{B}{N}\right)^{\sigma} < \frac{1}{\exp\left((\ln N)^{\alpha}\right)} B^{\sigma}.$$

(Note that the probability of each individual participant to get the prize in the lottery is $p = \frac{1}{N}$, and therefore $\frac{1}{p} = N$ in the denominator.)

This simplifies to:

$$\frac{1}{N^{\sigma}} < \frac{1}{\exp\left((\ln N)^{\alpha}\right)}$$

$$\exp\left((\ln N)^{\alpha}\right) < N^{\sigma}$$

$$(\ln N)^{\alpha} < \sigma \ln N$$

$$\frac{1}{\sigma} < (\ln N)^{1-\alpha}$$

$$\frac{1}{\sigma^{\frac{1}{1-\alpha}}} < \ln N$$

$$N > \exp\left(\frac{1}{\sigma^{\frac{1}{1-\alpha}}}\right) \approx 1711.60.$$

This means that up to a sample size of 1711, it is preferable to pay each participant an individual participation fee; starting from a sample size of 1712, it is preferable to enter participants in a lottery.

Bibliography

[1] Kahneman, Daniel, and Tversky, Amos (1979). "Prospect theory: An analysis of decisions under risk". Econometrica, 47, 313–327.

[2] Tanaka, Tomomi, Colin F. Camerer, and Quang Nguyen (2010). "Risk and Time Preferences: Linking Experimental and Household Survey Data from Vietnam." American Economic Review, 100(1): 557–71.

[3] Prelec, Drazen (1998). “The Probability Weighting Function.” Econometrica, 66(3): 497–527.

Chapter 12

The Problem with the Problem with Free Will

12.1 The neuroscientist's problem with free will

Cognitive neuroscientists have a problem with free will. In the folk sense of purely mental and not physical causation, we have come to agree that free will either does not make sense or does not exist. The reason we think we know this is because we have come to understand that all behavior comes from the brain. As an undergraduate, I regarded this as the single most important insight that neuroscience had ever come to: all behavior comes from the brain. It can adapt to environmental stimulation of course, and it is modulated by genes, random fluctuations of background neural activity and hormone levels, and its own history, in the sense that learning and experience can change synaptic weights and thus cognition and behavior. But nevertheless: all of these processes follow the laws of physics, and the brain is a deterministic system. Therefore there is little room in this picture for a non-physical entity which is causally responsible for how we think and act. The brain itself is plenty, thank you very much, we don't need souls to explain behavior.

This argument against free will is encountered in two incarnations: it is either formulated as a logical insight, to the effect that the concept of free will does not make sense; or it is presented as based on evidence. In the first, does-not-make-sense sense, the neuroscientific argument against free will is not based on experimental results; it is a premise, a point of departure for our experiments. It is therefore somewhat less interesting, and I will deal with it further down. The more interesting incarnation of the neuroscientist's argument against free will is the version which suggests that this insight is

based on experimental results. It is more interesting because it bites itself in the tail such that if it is true, then the same experimental results which prove the non-existence of free will also prove, ipso facto, that they themselves cannot be trusted. But let's take one step at a time.

On the surface, this evidence-based argument against free will is extremely compelling – in fact, it is more compelling than any other insight from cognitive neuroscience. The reason for this is that almost any result ever obtained in this field can be marshaled in support of the argument. Think about it: the point of cognitive neuroscience is to study the link between brain and behavior; and every study that finds another small link of this nature – some area lighting up in response to a reward, a jerk of the knee during transcranial magnetic stimulation, a change in impulsivity after hormone treatment – becomes yet another bullet in the arsenal of the neuroscientist who thinks that mental causation is just a load of romantic nonsense; and another blow to the romanticist's hope that maybe someday we'll find evidence of a behavior that doesn't have its basis in the brain.

What's more is that over the last few decades, the romanticist's hope should have been reduced considerably by the quality and sheer number of studies demonstrating physical causation of even complex cognitive processes. Using lesions and brain stimulation, these studies show that the behavioral consequences of altering brain activity are as pervasive as the supporting data are persuasive, ranging from perception to addiction, moral judgment, and economic decision-making. As early as the 1950s, Wilder Penfield showed that stimulating a patient's temporal lobe with electrical currents could evoke vivid memories (Penfield, 1952). In the 1990s, Bill Newsome and his colleagues conducted a series of groundbreaking studies in which they showed that electrical stimulation of area MT in the brain of macaque monkeys could bias the animals' perception of motion in a particular direction (Salzmann et al., 1990). More recently, an Iranian team around Arash Afraz, Roozbeh Kiani, and Hossein Esteky found that stimulating cells in the inferotemporal cortex of monkeys that encode faces could influence the monkeys' perception of noisy stimuli such that they became more likely to judge them to be faces (Afraz et al., 2006). In 2007, Naqvi and colleagues showed that smokers with lesions of the insula frequently lost their addiction to smoking, without relapse. The same year, Koenigs, Young et al. found that lesions of the prefrontal cortex caused humans to make more utilitarian moral judgments, e.g. judging it more permissible to sacrifice one human life to save several others. Using transcranial magnetic stimulation (TMS), Daria Knoch and her colleagues have shown that disrupting neural activity in dorsolateral prefrontal cortex diminishes subjects' ability to reject unfair offers and build a good reputation in economic exchange games (Knoch et al., 2006, 2009)

and make future-oriented choices (Figner et al., 2010). These are but a few examples; what they have in common is that they demonstrate an intimate link between neural activity and behavior, in particular, that neural activity is sufficient to produce even complex behaviors.

But the evidence-based argument against free will gets even stronger. A small group of neuroscientific experiments now deal head-on with the question of whether even conscious volition – i.e., free will itself – has a neural basis. In 1983, Benjamin Libet conducted what is probably the most famous of these experiments: he and his team asked subjects to move one of their fingers at will, whenever they felt like it. In addition, they recorded when each choice to move was made: in front of the subjects was a clock face with a rapidly rotating hand, and subjects were asked to remember the location of the hand at the instant in which they made the decision to move. This allowed Libet and his team to compare the timing of the decision to move with electrical activity in the subjects' brains measured with electroencephalography (EEG). Libet and his team found that the neural activity that initiated the movement in the brain, the "readiness potential", commenced several hundred milliseconds before the choice to move broke into subjects' awareness (Libet et al., 1983). Thus, the "free will" decision to move appeared to be not a cause of the eventual movement, but a consequence or an epiphenomenon, leaving little room for mental causation. A potential problem with this result is the fact that subjects' perception of the clock face might be biased – if there was some lag between their consciously experienced decision to move and the time it took them to judge the position of the rotating hand on the clock face, then the report of the decision to move could be delayed and therefore the decision itself might actually have coincided with, or preceded, the readiness potential. (mention Haggard here) However, a more recent study produced evidence for the original interpretation of Libet's finding. A team from Berlin told subjects to freely decide to move a finger on either their left or their right hand, at the time of their choosing (Soon et al., 2008). While subjects made these decisions, their brains were scanned using functional magnetic resonance imaging (fMRI). The researchers then used sophisticated algorithms called support vector machines (SVMs) to attempt to predict, purely based on neural activity, whether the subject would move their left or their right finger. The stunning finding of this study was that neural activity could not only correctly predict which finger the subject would move, but it could do so as early as ten seconds before subjects reported the conscious decision to move. Since the delay between the report and the actual decision is likely to be much shorter than that, this finding leaves little room for the alternative explanation of a lag in reporting the conscious decision. Thus, our brain appears to contain information about an

upcoming decision many seconds before we become consciously aware of it.

Together, these findings suggest not only that neural activity is sufficient to produce voluntary action, but in addition, that conscious volition may be an epiphenomon or a consequence, rather than a cause, of such neural activity. For the romanticist interested in preserving the notion of mental causation, this leaves little hope.

Thus, neuroscientists have a problem with free will in the sense of purely mental causation, and they have solid scientific evidence on their side – any study that ever showed a link between the brain and behavior, but in particular, studies that demonstrate that brain processes are sufficient to produce complex behavior, and others which show that conscious volition not only has a neural basis, but may be epiphenomenal to the neural processes governing an action. In addition, this argument gels perfectly with everything else we know about the brain – it has all the ingredients of a deterministic physical system, and there is neither room nor need to introduce mental causation. So, all behavior is determined, and neuroscientists are justified in having a problem with free will.

12.2 The problem with this problem

However, there is a problem with this problem. It is this: the neuroscientific experiments that lead us to think that free will in the sense of mental causation does not exist didn't conduct themselves. They were conducted by people with brains – brains which, if we believe the neuroscientific evidence and arguments, are just as deterministic and free of free will as all others. This means that "we" (that is, the conscious selves which like to dissociate themselves from the brain in an implicit form of dualism) have no choice in the experiments we conduct – they are the products of deterministic physical processes in our brains, and therefore they are completely predetermined. The hypotheses we come up with, the designs we choose, the analyses we perform, the interpretations we make, and the papers we write: our conscious selves have no say in any of it, all of these things were going to happen the way they did from the beginning of time.

I had said above that there was a second version of the neuroscientist's argument against free will, an "it does not make sense" version. In this does-not-make-sense sense, the non-existence of free will is an assumption of cognitive neuroscience, rather than an insight gained in experiments. The point of departure for cognitive neuroscience, this argument goes, already is that behavior comes from the brain; this does not first have to be proven in experiments. And again, because the brain is a deterministic physical

system, this leaves no room for free will. The problem with this view is that it, too, becomes its own victim: if free will does not make sense, then this argument does not make sense; whoever makes it had no choice in doing so, it was predetermined from the beginning of time that she would.

This insight instantly casts serious doubt on the findings of any experiment that has ever been done. For if the only laws that govern how these experiments are done are physical ones, not mental ones, then we no longer have much reason to think that our hypotheses are valid, our experimental designs are useful, our analyses appropriate, and our interpretations reasonable. Instead, our hypotheses, experimental designs, analyses, and interpretations will be those which our brain processes lead us to think are valid, useful, appropriate, and reasonable – we have no external criterion against which to assess whether this judgment is correct. Most damagingly, we have no reason to think that the results of these experiments will bring us closer to the truth; instead, we will think that they do, but again we have no criterion against which to assess whether this is so.

Wait, you say. Surely there are many criteria against which we can judge scientific experiments? Isn't a hypothesis valid if it is falsifiable? Isn't a design useful if it tests the hypothesis against a counterfactual? Most importantly, haven't we gotten closer to the truth if the theories we construct based on our experimental results make predictions that are accurate? And at the end of it all, can't we at least rely on logic, which dictates which conclusions are valid and which aren't?

If we accept the neuroscientific argument against free will, the answer to all of these questions is No. All of the criteria for progress that the philosophers of science have come up with over the centuries are, in the end, also just the products of these people's brains. Someone had to formulate them, others had to accept them. The fact that we adhere to some of them shows not that they are reasonable, but that our brain processes make our conscious selves believe that they are. The fact that not everyone agrees on which ones to adhere to shows not that there are different truths out there (how could there be?), but that individual differences in our brains make us disagree on which criteria are the right ones. Even logic is not a magic door to the truth – it, too, was formulated by human brains and thus falls victim to the same problem: logic may make perfect sense to us, but the fact that it does says more about what our brains are wired to find persuasive than what is true.

Of course it may be the case that our brain processes are wired such that they lead us to come up with experiments that actually do lead us closer to the truth. While this would be extremely unlikely to come about by chance (given that there are billions of possible experiments, the probability

of scientists conducting just the "right" ones is slim), one could imagine that evolutionary pressure may have equipped us with brain processes that are wired for "truth-finding". (Cite philosophy evolution argument from Putnam article "Why reason cannot be naturalized"). Note, however, that this argument rests on what we think we know about evolution by natural selection – another scientific insight that was had by a human brain! (The fact that it was Darwin's brain does not help much.) So we're back at square one.

In sum, the neuroscientist's problem with free will has a problem: it is self-defeating, in that if the claim that we have no free will is true, then that argument itself, and the evidence cited in its support, are ispo facto in doubt. But it gets worse. At this point, the reader will have noticed that the neuroscientific argument against free will refutes more than just itself: it leads to fundamental skepticism about any knowledge at all. The essence of the neuroscientist's argument is that we cannot trust our brains, and this is just the Cartesian Evil Demon argument reincarnate (cite something sensible here) – if we think our brains are deterministic, then we cannot know anything about the world at all, and we are thrown back onto thinking that we might be brains (or consciousnesses, rather) in vats. This epistemological dead-end has vexed philosophers for centuries, and now neuroscientists have found their own path into it, admittedly creative, but ending in the same place nevertheless.

So, this is where we are: if we believe the neuroscientist's arguments against free will, then we have to become fundamental skeptics about everything, including neuroscience itself. And thus we no longer have any reason to believe free will does or does not exist. Nor do we have reason to believe in anything else. Including this article.

Bibliography

[1] Figner B, Knoch D, Johnson EJ, Krosch AR, Lisanby SH, Fehr E, Weber EU. "Lateral prefrontal cortex and self-control in intertemporal choice." Nature Neuroscience (2010) 13, 538–539

[2] Knoch D, Pascual-Leone A, Meyer K, Treyer V, Fehr E. "Diminishing reciprocal fairness by disrupting the right prefrontal cortex" Science (2006) 314, 829–832.

[3] Knoch D, Schneider F, Schunk D, Hohmann M, Fehr E. "Disrupting the prefrontal cortex diminishes the human ability to build a good reputation". Proceedings of the National Academy of Sciences of the USA (2009) 106, 20895–20899.

[4] Koenigs, M.*, Young, L.*, Adolphs, R., Tranel, D., Cushman, F., Hauser, M., Damasio, A. (2007). Damage to the prefrontal cortex increases utilitarian moral judgments. Nature, 446, 908-911.

[5] Libet, B.; Gleason, C.A.; Wright, E.W.; Pearl, D.K. (1983). "Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential). The unconscious initiation of a freely voluntary act". Brain 106 (3): 623–642.

[6] Penfield, W. (1952). Memory Mechanisms. AMA Archives of Neurology and Psychiatry 67, 178-198.

[7] Salzman, CD, Britten, KH and Newsome, WT (1990). Cortical microstimulation influences perceptual judgments of motion direction. Nature 346: 174-177. pdf

[8] Soon CS, Brass M, Heinze HJ, Haynes JD. Unconscious determinants of free decisions in the human brain. Nat Neurosci. 2008 May;11(5):543-5.

Part III

Appendix

List of Figures

List of Tables